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CONTENTS.

PAGE

On Obliteration of the Superior Vena Cava. By WILLIAM OSLER, M. D., ... 169

A New Method of Hemo-Alkalimetry and a New Hemo-Alkalimeter. By ARTHUR DARE, M. D., ... 175

John Collins Warren. By J. G. MUMFORD, M. D., ... 180

PAGE

The Etiology of Serofibrinous Pleurisy, with Reference to Cytological Diagnosis. By C. H. BUNTING, M. D., ... 185

A Case of Peptic Ulcer in the Jejunum of a Dog following Gastro-enterostomy, with a Review of the Cases Reported in Man. By S. H. WATTS, M. D., ... 191

The Advantages of the Sims Posture in Cystoscopic Examinations. By JOHN A. SAMPSON, M. D., ... 194

ON OBLITERATION OF THE SUPERIOR VENA CAVA.

BY WILLIAM OSLER, M. D.,

Professor of Medicine, Johns Hopkins University.

While signs of compression of the superior vena cava are not very uncommon in cases of aneurism of the aorta and in mediastinal tumors, instances of complete obliteration of the vessel, with the establishment of collateral circulation, are extremely rare. I here report one case which was under our observation for nearly three years, long enough to make the diagnosis of fibroid obliteration by exclusion, and a second in which the obliteration was due to compression in Hodgkin's disease. A third case due to aneurism I have already reported.[1]

Dr. Hume has collected for me from the literature the histories of 29 cases of complete obliteration of this vessel. Many of the reports are imperfect, and only the anatomical record is given. Of the cases 13 were males, 12 females and in 4 the sex was not specified. Eighteen of the patients were between the ages of 30 and 60. So far as the cause could be ascertained the cases could be grouped as follows: I. Thrombosis due to disease within the vein, 10 cases. Of these, 8 seem to have been due to a simple phlebitis; one, the case of Duchek, was a propagated thrombus from the periphery, and one a remarkable case of tuberculosis endophlebitis (Banti). By far the largest number of cases were due to: II. Disease outside the vein, 19 cases, grouped as follows: (a) tuberculosis, 4 cases; (b) mediastinitis, 4 cases; (c) aneurism, 4 cases; (d) syphilis, 3 cases; (e) periaortitis, 2 cases; (f) carcinoma, 1 case; (g) fibroma, 1 case.

The symptoms of the condition depend entirely upon the degree to which compensatory circulation has been established. Obliteration of any one of the three great veins of the body may exist for many years with even good health and a completely effected collateral circulation. There seem to be two groups of cases, one in which the patient has had for years complete compensation and good health and the symptoms set in acutely. This was well illustrated in a case at the Montreal General Hospital, reported by Wilkins (Case 6), in which I made the dissection. The patient was a robust, hale man, aged 34, who twelve months before, while lifting, felt something give way. He had occasional attacks of dyspnœa and a smothering feeling. His urgent dyspnœa came on somewhat suddenly, and for three months he had a great deal of

[1] Journal of the American Medical Association, June 7, 1902.

oppression of breathing, due in large part to recurring effusion into both pleural sacs. There was complete fibroid obliteration of the superior vena cava.

In the second group the symptoms of obstruction of the venous circulation are constantly present, though varying in intensity, as in the case here reported, and the condition may be consistent with a fair measure of health.

CASE I.—*Clinical Summary: Hard work, alcohol and exposure; dyspnœa, swelling of the neck and face; gradual distension of the superficial thoracic and epigastric veins; improvement for a time; gradual increase in the size of the superficial veins; on third admission tubercle bacilli were found; final admission with fever and delirium and unconsciousness; lumbar puncture, tubercle bacilli in exudate; tuberculous caries of spine; fibrous mediastinitis with obliteration of the superior vena cava and innominate veins; tuberculous meningitis.*

Charles Diggs, colored, butcher.

First admission was on Dec. 7, 1898. The patient was at this time 22 years old. He complained of tightness across the chest, dyspnœa and swelling of the neck.

The family history was unimportant.

Personal History.—Patient had had no rheumatism, typhoid fever, malaria or pneumonia. He had not been subject to headache, nor had he any sensory disturbance. Venereal history; gonorrhœa six times. Patient says he has had a chancre, but from the description the sore was probably chancroid, and there have been no secondary symptoms. Tobacco: patient began smoking at eight years of age, and smokes several packages of cigarettes a day. Alcohol: gin, whiskey and beer have been used abundantly, fifteen glasses of beer at least a day. He has often been drunk. He is a heavy eater. He is often exposed to the weather, and has a great deal of heavy work.

The present illness began six days before admission. The patient went to bed feeling well, and woke up with dyspnœa. He noticed that his neck was swollen, and felt as if something were pressing against his chest tightly. The pain in his chest was not definitely localized. He noticed that his face was flushed. The patient had not been drinking on the night of the first symptoms, but had been drunk the previous night. The condition had grown worse each day. He had been treated before admission with doses of nitroglycerin, 1-100 of a grain, and had been given irrigations for his urethritis. The appetite was good and his bowels were regular. He thinks he has lost ten pounds since the onset of the symptoms.

Present condition.—The patient is a strong, well-nourished mulatto. The face and eyelids are puffy. The tongue is coated with a yellowish fur. No anæmia of the mucous membranes. The neck is full; the vessels are dilated and distended with blood. Slight general pulsation of the neck. No tracheal tugging. The chest is well formed; respiratory movements equal. There is distinct tenderness on pressure in the epi-sternal notch. The right clavicle is more prominent than the left.

The lungs are clear throughout on auscultation and percussion. There is no dulness below the sternum suggestive of mediastinal growth. Superficial veins on the thorax and of the upper right arm and shoulder are dilated.

Heart not enlarged; sounds quite clear. Pulse 78 to the minute, of fair volume and tension. The right radial is slightly fuller than the left; the vessels are palpable.

Glands: The inguinal, the left epitrochlear, the posterior cervical and the submaxillary are enlarged. There is no œdema of the legs. The reflexes are apparently normal. The differential count shows a practically normal ratio of leucocytes. The X-ray picture was negative and nothing abnormal could be seen with the fluoroscope.

Two weeks after admission a slight pleuro-pericardial friction rub was noted. On December 21 the patient was discharged, distinctly improved. The veins of the neck were less distended than on admission. During the first three days he had had a slight temperature, but since that time had been practically free from fever.

The patient was admitted a second time on December 1, 1899, complaining of pain in the chest and of swelling of the face. Since leaving the hospital he had been unable to do any hard work. Every exertion would cause swelling of the face and neck. He had been "drinking and sporting" until two months before the present admission, and during these two months there had been a steady pain in his chest, sometimes extending into the arms. He had slept poorly. On examination the superficial veins were found dilated in the forearms as well, and the thoracic subcutaneous veins were found to anastomose with the superficial and deep epigastrics. The current in these thoracic veins was distinctly from above downwards. The radial pulses were equal. No thrill and no diastolic shock were observed during his stay in the hospital. The patient was discharged improved on January 2, 1900, again with a diagnosis of probably intra-thoracic tumor.

On several occasions during the spring of 1900 the patient was demonstrated to the students at the out-patient clinic. The absence of all signs of aneurism and of enlargement of the glands, the negative character of the X-ray picture, the slow course of the disease, led to the diagnosis of fibroid obliteration of the superior vena cava.

He was admitted for the third time on June 20, 1900, complaining of pain in the chest and in the right arm and of a cough. While away from the hospital he had been comfortable so long as he took care of himself. The cough had lasted three weeks and had been accompanied by much expectoration. The right radial pulse was now distinctly fuller than the left. The veins were more dilated on the right side of the thorax. The right lung was distinctly impaired. Tubercle bacilli were found on June 23. The expectoration was muco-purulent in character and very foul. There was a great deal of insomnia due to the cough. The patient insisted upon

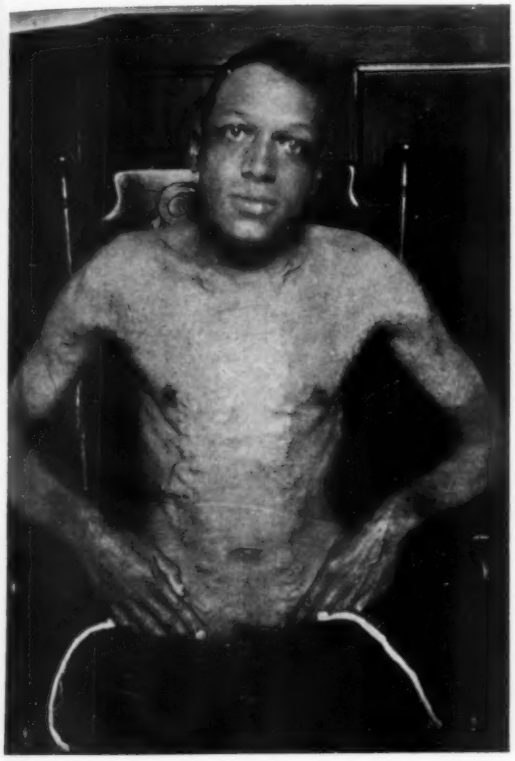


FIG. 1.

leaving the hospital on June 25. Fig. 1 shows the distension of the superficial veins.

The fourth and last admission was on February 16, 1901, when the patient came in complaining of cough which had lasted three weeks. He had been spitting blood at intervals for a year and had night sweats. His appetite was poor, and he had had a good deal of vomiting. He had also had constipation. Two days after admission tubercle bacilli were again found, and the condition of the right lung was worse than on the previous admission. His cough was racking and very severe. On February 31 he became noisy and profane and refused to keep on his clothes, and during the afternoon was found lying on the floor, and irregular convulsive movements of the extremities were noted, and the patient was apparently unconscious. The temperature rose to 102.6°. On being put to bed the patient kept making peculiar grimaces, but soon became rational and did not remember the period of unconsciousness. His speech was distinctly thick like that of a drunken man. On February 24 the patient became restless; there was marked cyanosis of the face and extraordinary injection of the conjunctivæ; there was definite nystagmus; the pupils were unequal, the left being larger; there was frothing at the mouth. The dilatation of the veins had become appreciably greater. Later in the day the cyanosis increased. Bleeding was resorted to and temporarily quieted the patient. The respirations were very stertorous throughout the day. Soon after midnight, after a violent convulsive attack, the patient became quieter and died at 1.50 a. m. on February 25.

Lumbar puncture was done at the time of death and the fluid showed leucocytes and tubercle bacilli.

Autopsy by Dr. MacCallum on February 25, 1901, nine hours after death.

Anatomical Diagnosis.—Chronic tuberculosis; tuberculous caries of spine; fibrous tissue growth in the adjacent regions, with involvement and occlusion of vena cava superior and of innominate veins; establishment of extensive collateral venous circulation; tuberculous meningitis.

The body of a young negro, 170 cm. long. Over the anterior thoracic and abdominal regions, reaching down to the inguinal regions, there are many tortuous and distended subcutaneous veins. To facilitate the dissection of the venous system hot water and then colored wax were injected into the femorals. The dissection reveals the following condition of the venous system: The right jugular and the right subclavian, much dilated, formed the right innominate, which is immediately obliterated to form a dense thick cord. The right internal mammary is obliterated at its junction with this innominate. The left internal jugular is obliterated, or partly so, in the neck. It joins the left subclavian to form again a fibrous mass which represents the innominate on the left side. The two innominates unite to form a fibrous mass which represents the superior vena cava (Fig. 2). Within the pericardium, however, this vessel is patent from the point where it receives the wide azygos vein to the heart, but it is not more than 15 mm. wide and its walls are quite thick. The azygos vein is 1 cm. in diameter, and there is no obstruction at its mouth. The anterior perforating branches of the internal mammary are very wide and connect with the long tortuous subcutaneous veins. The superficial brachial veins communicate similarly with these tortuous veins, forming a pre-pectoral anastomosis (Fig. 3). The tortuous veins communicate with the superficial and probably with the deep pectoral veins in the inguinal region. The sinuses in the cranial cavity are dilated and contain non-adherent thrombi. The left jugular vein being occluded, blood from the head must have passed out mainly by way of the right jugular, through the right subclavian, through the pre-pectoral anastomosis with the anterior perforating branches of the internal mammary and with the subcutaneous tortuous veins to the superficial epigastrics and thence to the inferior vena cava, which was normal throughout its course. An alternate course would have been from the subclavian to the upper right intercostal veins through the perforating branches of the latter and thence to the azygos, which was very large (12 cm. in diameter). On the left the blood from the subclavian vein followed a similar course. The obliterated portions of the innominate and jugular veins lie as a firm mass of fibrous tissue firmly adherent to the spinal column. Peritoneum and pericardium normal.

Lungs: Both adherent to the pleura at the apices; bronchial glands enlarged; considerable œdema present. Many tubercles at each apex and throughout the lung. A cavity 2 cm. in diameter present in the right lung. Between the tubercles the intervening lung is scarred, but there is no pneumonia. Both lungs are adherent to the vertebral column at their upper portions.

Spleen: Substance is pale and flabby.

Liver: Near the edge there is a puckered white opaque thickening of the capsule.

Stomach, pancreas, kidneys, testes normal.

Vertebræ: Removal of the fibroid mass surrounding the innominate veins reveals an erosion of the centra of the last cervical and the first two or three thoracic ventebræ, with complete denudation of the bone. There is a focus of disease in the last cervical and first thoracic vertebral centra. These have collapsed and caused a slight skoliosis and possibly some kyphosis. The cavity thus formed extends into the spinal canal but causes no special compression of the cord. The adjacent centra are much eburnated. The fibrous tissue lying upon this cavity in the vertebræ is continuous with that about the innominate veins. There is no marked abnormality in the cranial vessels. The pia, especially in the fissure of Sylvius about the cerebellum, the base of the brain and the medulla, shows many miliary tubercles. The lateral ventricles are slightly distended with fluid. The ependyma shows a fine granulation.

CASE II.—*Hodgkin's Disease; Compression of Superior Vena Cava; Extensive Collateral Circulation; Formation of Phleboliths; Unusual Chronic Course; Autopsy.*

M. H., aged 31, varnisher, applied at the Medical Dispensary, October 4, 1889, complaining of swelling of the neck and of the face. Family history is good. None of his relatives have had any glandular enlargements.

He himself has always been well and strong, with the exception of the usual diseases of childhood.

About 1884 he first noticed swelling of the glands of the neck, chiefly on the right side. It did not increase much, but shortly afterwards he noticed the glands on the left side were a little large. They, however, did not interfere with his work. Throughout the year 1887 there was a decided increase in the glandular enlargement, and about Christmas of that year the face was a good deal swollen and the veins of the face and of the arms and front of chest began to swell. He also

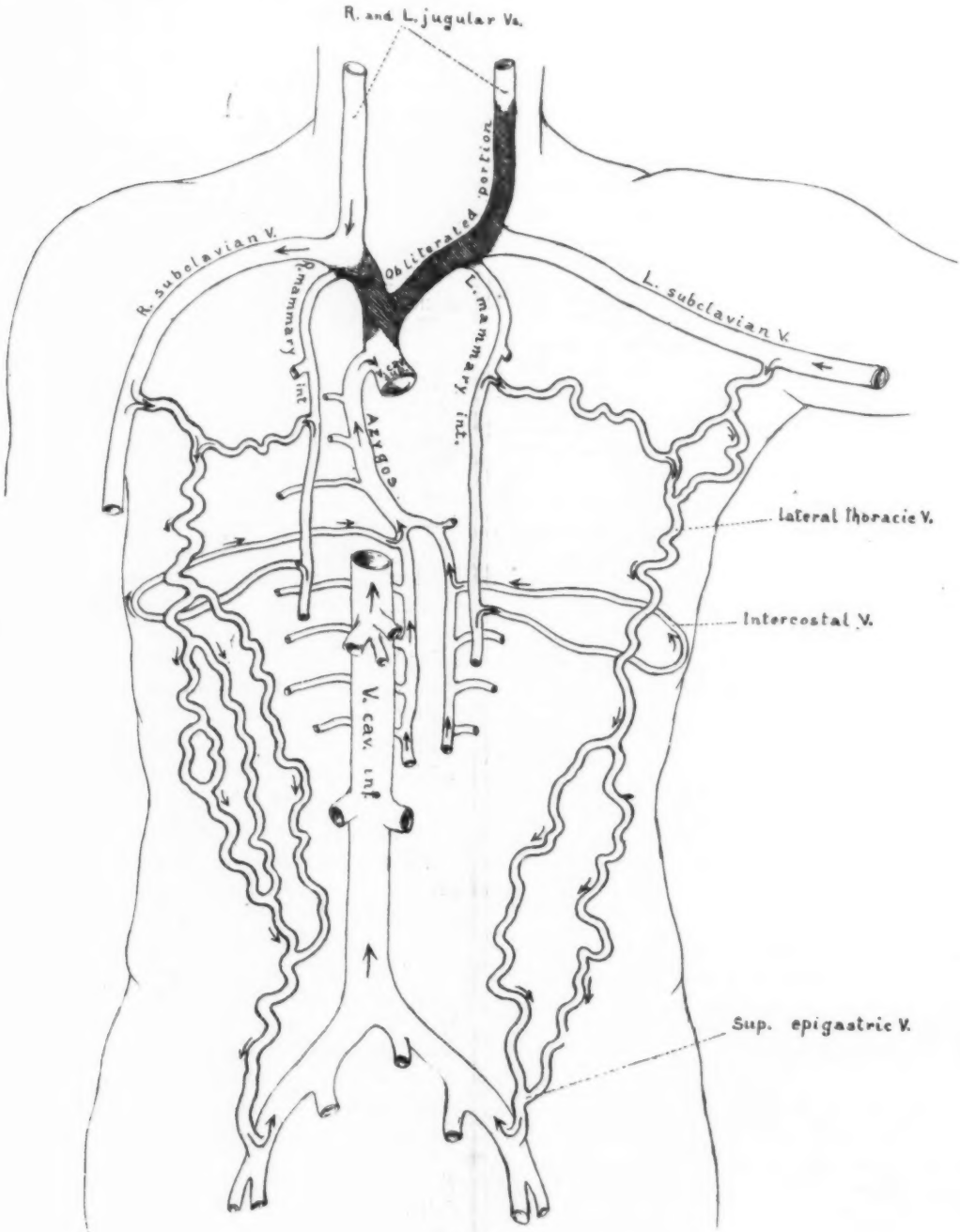


FIG. 3.

FIG. 2.

had shortness of breath on slight exertion. Through last year and the present year this condition has persisted. The glandular enlargement has been progressive; the swelling of the veins very marked, and on exercise he gets extremely livid.

On October 4, 1889, I dictated the following note: The patient is a small, spare man; the face is greatly suffused and swollen, the conjunctiva moist and injected, and about the lips and cheeks there is slight lividity. The tongue also is somewhat cyanosed. The venules are prominent and the veins of the cheeks and forehead are distinct. There is great enlargement of the glands of the neck, particularly on the right side. They are firm and hard, extending on both sides of the sterno-mastoid muscles and reaching to the clavicles, the outlines of which are obliterated by the swelling. The axillary glands are not enlarged. When stripped the most striking feature is the enormous distension of the thoracic and abdominal veins. The manubrium looks prominent and the first and second interspaces near the sternum look full. Beneath the skin of the sternum and over the whole front of the thorax large veins can be seen, and on palpation there is felt a soft plexus of distended vessels. Palpation causes great pain over the manubrium and the first and second left interspaces.

On percussion there is absolute flatness on the manubrium, extending into the infra-clavicular space and as low as the nipple, where it is continuous with that of the heart. The dulness extends only about two inches from the sternal margin. The apex beat is neither visible nor palpable. Percussion is clear on the right side in front and on both sides behind.

On auscultation, the heart sounds are clear, the aortic second accentuated; no murmur in aortic region or at apex. Respiration is very feeble and distant in left infra-clavicular and mammary regions. The radials are equal. There is no lividity of the hands; no enlargement of the veins; no clubbing of the fingers.

The abdomen looked full and beneath the skin there could be seen the superficial epigastric veins enormously distended; the right one almost as large as the index finger. The blood current in them is from above downwards.

The patient was placed upon Fowler's solution, minims v, to be increased. From this time until his death, 1893, that is for fully three years and a half, this patient was under our observation, and I showed him repeatedly at the clinics, and once at the Hospital Medical Society.

I will note here some of the special features in his case as dictated by me at different periods.

December 7, 1889, venous engorgement not so marked. He has had two smothering attacks at night and has had blood spitting.

January 18, 1890, he was better, the glandular enlargement was not so marked, eyes not so suffused. He has been taking Fowler's solution of arsenic up to 15 minims three times a day and has had a little diarrhœa. The swelling of the glands has distinctly lessened. He has a good appetite.

February 25, 1890, the note is: Feels very well; has improved wonderfully; scarcely any more swelling on the right side than on the left; the glands of the lower part of the neck are still enlarged, and only slightly tender. Difference in the pulses still apparent.

March 24: Glandular enlargement even smaller than before; less tender. He has had a distressing papular eruption on the face, hands and neck, which itches very much. Throughout the summer he improved a great deal and felt very much stronger.

On September 4 I dictated the following note: There is a red papular rash which itches. The swelling on the right side of the neck is still painful to the touch. Though the swelling in the neck has diminished, the cervical glands are still enlarged; the distension of veins over the front of the chest is now enormous, extending on either side to the nipple line; the episgastric veins are also enormously enlarged and plexiform. In some of the larger veins over the manubrium thrombi are to be felt. The large cutaneous veins extend over the right shoulder and biceps. The face is still suffused, but not nearly so much as before. The dulness persists over the manubrium and the left infra-clavicular space. Heart sounds are clear; there is no murmur; no venous hum. There is very marked difference between the breath sounds in the left and right infra-clavicular regions; in the former they are scarcely audible.

Throughout the year 1891 he was frequently under observation and there was very little change. I examined the heart repeatedly. There was never any venous hum, never any murmur. He took the Fowler's solution at intervals; towards the end of the year he had some pain in the left arm and the left side of the chest.

On April 5, 1892, I made the following note: General condition remains unchanged, though the glands of the right side of the neck are now not very much swollen. They are well marked on the left side. The sternum is perhaps more prominent, but it is difficult to say whether this is due to increase in the bone itself or to the enormous veins in the subcutaneous tissue. The skin is not abraded or reddened. The plexuses of veins already referred to is very marked. Just under the left nipple one of the larger veins contains a thrombus and higher up towards the manubrium there are several phleboliths the size of peas. The right epigastric vein is still larger. There is a slight heaving of the whole chest with each cardiac impulse. The heart sounds are clear, aortic second not accentuated. No venous hum over the plexus. Face a little suffused; no change in the pulmonary condition.

In July the patient had a hemorrhage, stated to be from the lungs. In September he said that he had been fairly well all summer and had tried to do a little work. The face was

congested and full; the glands on the right side of the neck still considerably enlarged. The superficial epigastric veins were distinctly smaller, but there seemed to be no change in the condition in those of the sternum. Early in January I showed the patient at the clinic. The condition was practically the same, though the epigastric veins were certainly not quite so large.

The patient died in March, 1893, and an autopsy was obtained by Dr. Flexner, but under most unfavorable circumstances, and the thoracic organs had to be hurriedly examined, so that no complete dissection could be made in situ. The superior cava was completely obliterated by the enlarged mediastinal glands.

Summary of Cases.

(Cases from the literature of fibroid obliteration of the superior vena cava, collected by Dr. E. H. Hume.)

A. Thrombosis due to disease within vein.

I. (a) Phlebitis.

1. Breschet: Traité des maladies des artères, &c., Paris 1819 (translation of Hodgson's Diseases of Arteries), No. 150; Preparation in Museum of Faculty of Medicine, Paris; a wax model made under direction of Dupuytren. Thrombosis of vena cava superior.

2. Wolff: Mag. f. d. ges. Heilk., Berlin, 1823, XIV, 570. Female, 19; sarcoma of shoulder. P. M., inflammation of right auricle and pericardium; thrombosis of vena cava superior.

3. Claverie, G. E.: Thèse de Paris, 4°, 1858. Male, 52, jeweler; onset of symptoms gradual, lasting thirty years; gout three years previously; death from gangrene of foot following gout. P. M., vena cava a fibrous cord; pericardium adherent; lungs, chronic phthisis.

4. Rees: Lancet, 1860, II, 585. Female, 48; œdema of upper and lower extremities. P. M., complete obliteration; thrombosis of vena cava superior, evidently phlebitis; disease evidently propagated from right auricle.

5. Rees: Guy's Hospital Reports, 1861, 3. s., VII, 113. Female, 54; heart disease. P. M., entrance to vena cava superior obliterated by phlebitic thrombi.

6. Wilkins: Lancet, 1883, I, 812. Male, 34, naval officer; strain twelve months previously. P. M., thrombosis with complete obliteration of vena cava superior.

7. Garcia Rijo: Crón. méd. quir. de la Habana, 1887, XIII, 412. Male, 38, soldier. P. M., complete obliteration of vena cava superior by thrombosis.

8. Hirschlaff: Inaug. Diss., Berlin, 1893. Female, 51; diphtheria at eighteen; rheumatism at twenty. P. M., mitral stenosis; aortic and mitral insufficiency; marantic thrombosis of vena cava superior, with complete obliteration.

(b) Propagated Thrombus.

9. Duchek: Prager Vierteljahrsschrift, 1854, XLI, 109. Pathological specimen in Museum at Vienna. No clinical history. Vena cava superior obliterated by thrombus propagated from periphery.

(c) Tuberculous Endophlebitis.

10. Banti: Sperimentale, Mem. Orig., Firenze, 1891, XLV, 408. Male, 46; carpenter; death from acute miliary tuberculosis. P. M., vena cava superior a hard cylinder; a vegetative mass occupying the auricular entrance to vena cava superior; microscopically, tuberculous endophlebitis.

B. Thrombosis due to disease without the vein.

I. Tuberculosis.

11. Tonnelé: Jour. Hebd. de Méd., 1829, V. Male, 2; cough, diarrhœa, vomiting. P. M., complete obliteration; encysted tuberculous mass, caseous at centre, connected vertebræ to vena cava superior and obliterated the latter; thrombosis of superior longitudinal sinus propagated to vena cava superior.

12. Reid, J.: Edinb. Med. & Surg. Jour., 1835, XLIII, 297. Female, 40, of irregular habits. P. M., vena cava superior a cartilaginous cord connected to right bronchus. Process probably originated in tuberculous bronchial glands.

13. Paulus: Oesterreich med. Woch., 1842, 2 quartal., No. 14. Clinical history not given. P. M., secondary caries of right petrous bone; complete obliteration of vena cava superior by propagated thrombus.

14. Duchek: Loc. cit. Male, 52. P. M., vena cava superior obliterated and attached by fibrous tissue to right bronchus. The process evidently originated in a tuberculous gland.

II. Mediastinitis (unclassified).

15. Oulmont: Mem. Soc. Méd. d'obs., Paris, 1856, III, 391, 468. Female, 49, cook. P. M., a fibrous mass in mediastinum attaching bronchial gland to left bronchus and vena cava superior; vena cava superior completely thrombosed.

16. Habershon: Lancet, 1875, II, 837. Male, 37, coalheaver. P. M., complete obliteration; chronic mediastinitis leading to fibrous transformation of vena cava superior; evidently congenital, certain valvular defects being present.

17. Williams: Proc. Path. Soc., Dublin, 1877, n. s. VIII, 8. Female, 27; rheumatism eight years before. P. M., vena cava superior a fibrous cord, probably due to inflammation around mediastinal glands.

18. Roberts: Lancet, 1893, II, 1386. No clinical history.

P. M., complete obliteration; fibrous transformation of vena cava superior, probably due to mediastinitis.

III. Aneurism.

19. Martin-Solon: Arch. de Méd., 1836, 2. s., X, 296, Female. P. M., enormous aneurism of aorta; vena cava superior obliterated in walls of sac.

20. Duchek: Loc. cit. Male, 39. P. M., vena cava superior obliterated and lost in the walls of an aneurismal sac.

21. Watson: Practice of Physic, Ed. Condie, 1866, 798. Male, 33. P. M., complete obliteration, vena cava superior being lost in walls of sac of an aneurism of aorta.

22. Russell: Medical Times and Gazette, 1871, II, 130. Male; injury to chest three years previously. P. M., complete obliteration; vena cava superior lost in walls of sac of huge aneurism of aorta.

IV. Syphilis.

23. Willigk: Prager Vierteljahrsschrift, 1853, XXXVIII, 20. Female, 44, laborer. P. M., complete obliteration; fibrous vena cava superior included in syphilitic scar tissue in right bronchus.

24. Duchek: Loc. cit. Female, 47. P. M., Vena cava superior obliterated and attached by fibrous scar tissue, evidently syphilitic, to right bronchus.

25. Fraenkel: Deutsche Med. Woch., 1891, XVII, 1378. Male, 45, history of syphilis and alcoholism. P. M., complete obliteration; compression of vena cava superior by mediastinal syphilitic granuloma; thrombosis of vena cava superior.

V. Periaortitis.

26. Rigler: Wien Med. Woch., 1858, VIII, 1. Male, 70; painter; rickets and typhoid; acute rheumatism often after 28. P. M., dilatation of aorta; periaortitis; thrombosis of vena cava superior.

27. Meigs: Transactions of the Coll. of Phys., Phila., 1886, 3. s., VIII, 13. Male, 72; general arterio-sclerosis; sudden vertigo on day of admission. P. M., complete obliteration; mediastinitis and periaortitis, leading to thrombosis of vena cava superior.

VI. Carcinoma.

28. Barth: Bull. soc. anat., Paris, 1853, XXVIII, 4. Female, 36, healthy, 5-para. P. M., cancer of lung and heart; vena cava superior obliterated by organized clot from the auricle upwards.

VII. Fibroma.

29. Pastau: Virch. Arch., 1865, XXXIV, 236. Female, 42. P. M., fibroma pressing on innominate artery and vena cava superior; complete thrombosis of vena cava superior, with calcification of walls.

A NEW METHOD OF HEMO-ALKALIMETRY AND A NEW HEMO-ALKALIMETER.[1]

BY ARTHUR DARE, M. D.

Numerous methods have been devised from time to time for the purpose of studying the concentration of alkali in the blood; most of these devices employ litmus, lachmoid, erythrosin, or some other one of the chemical indicators, and require for their application a volume of blood only procurable by the scalpel or cupping-glass.

While results fairly approximate may be obtained with color indicators they are not sufficiently accurate to determine the delicate variations required to render the results of value, owing to the fact that laked blood placed upon alkalin (blue) litmus or lachmoid paper, appears red from the presence of hemoglobin and not unlike that of acid litmus; hence the danger of confusing the two phenomena.

As an illustration of the inaccuracies of tests based upon the appreciation of vague colors, we may take our experiment of titrating the blood with tartaric acid. As the reagent is added cautiously, the color intensity is reduced by dilution, but the brilliancy of the hemoglobin is retained; but when the basic principal of the blood is entirely neutralized, the pink color of diluted hemoglobin changes to a straw yellow. This color change, however, cannot be observed in spleno-medullary leukemia, owing to the turbidity of the blood occasioned by a great leukocytosis, the refraction of the leukocytes obscures the brilliant red of the hemoglobin, and the blood and water mixture appears milky rather than red.

I desire to present for consideration a means for determining the neutralization of the blood at an exact and definite point; a method free from the inaccuracies of test papers prepared chemically by an excess of acid or alkali above the neutral point, and one that does not react solely to certain of the complex ingredients of the plasma but shows the point of neutralization of the whole blood.

Blood when examined with the spectroscope gives the characteristic spectrum of oxyhemoglobin, unless changed by the absorption of certain gases or the introduction of substances that modify the composition or functional conditions of the plasma. Under these conditions, the blood spectrum may be altered to that of methemoglobin, carbonyl or carbon monoxide hemoglobin, etc.; but the presence of hemoglobin is still evident by the characteristic spectra. In the case of oxyhemoglobin two dense shadows appear in the highly colored field of the spectroscope, as parallel absorption bands between D and E of Fraunhofer's lines, the line nearest to D is darker, narrower and more strongly marked; that next to E is less sharply defined and broader.

[1] Read before the Johns Hopkins Hospital Medical Society, April 6, 1903.

As the blood is diluted and examined spectroscopically in a stratum of a certain thickness, the absorption bands of oxyhemoglobin are observed to fade away, but are made to reappear when this stratum thickness is increased. When, however, blood is titrated with an acid reagent, the spectrum of oxyhemoglobin disappears and cannot be restored by the addition of an alkali or by increasing the stratum to the maximum, which is capable of illumination for examination spectroscopically, hence the spectrum of oxyhemoglobin may be removed by dilution or by neutralization, in the latter case the hemoglobin is changed and its characteristic spectrum is destroyed permanently, while in the former it can be made to reappear by increasing the concentration of the hemoglobin.

Upon this observation our method is based, yet it will not modify the results as an indicator whether the absorption bands of oxyhemoglobin disappear at the exact point of neutralization or beyond, as it requires a definite amount of acid to cause their disappearance, as can readily be shown by the experiment of adding alkalies or acids to the blood mixture under examination and again titrating, the result being anteceded or delayed by the increase or decrease of alkalinity artificially modified. Therefore we have in the blood *a natural indicator* observed with the spectroscope up to the exact point of departure, which we assume is the point of neutralization; but which cannot be verified by the usual chemical procedures owing to the fact that blood does not give an alkalin reaction to litmus, lackmoid or other color indicators, unless some procedure is introduced that sets the alkali free from its combination in the plasma.

The hemoglobin concentration of the plasma would possibly determine the point of neutralization (or decolorization of the hemoglobin), were all specimens of blood of uniform reaction, but as a certain amount of basic material is to be neutralized before the hemoglobin becomes decolorized, the point of departure is dependent upon the alkalin principle, rather than the quantity of the hemoglobin.

The combination of acid with the basic principles of the blood probably generates carbon-dioxide, which has been thought to decolorize the hemoglobin, but experiment teaches that this is not the case. A blood and water mixture subjected to the direct and continuous flow of carbon-dioxide for an hour failed to modify the alkalinity or to change the color concentration in the slightest degree, hence the carbon-dioxide formed by combination in no wise influences the uncombined principles of the plasma, which are only neutralized by the direct action and combination of the acid reagent.

The final result of volumetric estimation is more precise when a large volume of acid reagent is employed for neutralizing the basic principles of the plasma, permitting as it does of a wider range of measurement and therefore of more conclusive deductions.

In the method suggested by Engel, normal blood is neutralized by 0.5 cc. of acid reagent while our method requires 2.0 cc. for the same purpose, hence Engel divides his result by five while we divide by twenty; naturally the latter method possesses the essential advantage of determining slight variations. When desirable, the result can be still further divided by titrating with a reagent one-half the strength as the one employed; namely, 1/200 of the normal.

Advantage not only to the patient, but also to the hematologist, is derived from the quantity of blood required for an observation. Only one-fourth the quantity of blood necessary for Engel's instrument is needed for an application, as but one small drop is required, while the capacity of Engel's pipette is so great that a drop sufficiently large will rarely adhere to the skin and without assistance it will be necessary to reapply the pipette; this is especially true of profound anemia, when it is often difficult to obtain enough blood for a complete study of the same specimen or to obtain a proper sized drop owing to the lessening of the viscosity of the plasma.

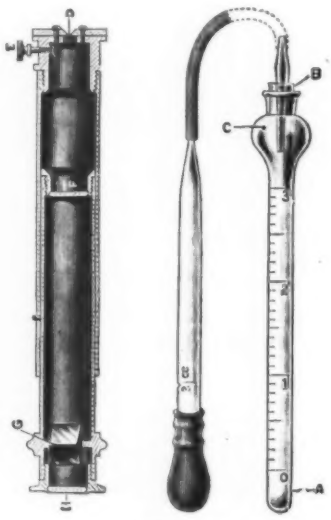


FIG. 1.

Hemo-alkalimeter about one-half actual size. A. Alkalimeter tube. B. Automatic blood pipette. C. Opening for the admission of air. D. Cleft of spectroscope, into which the alkalimeter tube is placed at the point of A. E. Cleft adjustment. F. Diaphragm to intercept reflected light. G. Nicol prism. H. Eye piece.

In selecting a reagent, we were influenced entirely by the merits of various acid reagents; rather than the results of combination with the complex and doubtful composition of the blood.

The concentration of acid in solutions of acid phosphoric varies, and standardization is necessary whenever it is employed, owing to its affinity for water.

Oxalic acid contains a greater amount of water of crystallization and hence is more efflorescent than our reagent, acid tartaric, which contains no water of crystallization and is therefore more uniform in quantivalence value. The mineral acids while probably more permanent in aqueous solutions are open to the objection that the concentration varies to such an extent that they require standardization whenever a reagent is to be prepared.

Description of Instrument.—The hemo-alkalimeter is made up as follows: The first part (Fig. 1, B) consists of a glass stopper through which passes an automatic capillary blood pipette containing 20 cmm. by volume or 15 mgs. by weight (of normal blood), the exposed end of which is ground to a tapering point. The caliber of this little tube remains the same throughout. It may be remarked that a thick glass is used in the manufacture and presents the advantage of being so ground as to afford a magnifying surface, in this respect resembling the clinical thermometer. This stopper and the contained blood pipette are fitted into a clear glass test tube (Fig. 1, A) containing 3 cc., the upper end of which is expanded into a bulb containing on its side a minute opening (Fig. 1, C) for the purpose of admitting air. The top of the bulb is placed close to the stopper, allowing the capillary tube to pass to the center of the bulb, this situation being selected as preventing an adherence of the blood between the sides of the test tube and the capillary pipette. The test tube is graduated in cubic centimeters, and the equivalent in mgs. of NaOH to 100 cc. of blood also represented.

The second part of the instrument, *the reagent pipette*, consists of an appliance made of a glass tube terminating with a rubber bulb and having at its other extremity a piece of rubber tubing which fits over the sharpened end of the capillary blood pipette previously described. In conjunction with this instrument, a spectroscope should be used, the Browning pocket spectroscope answering all purposes.

The test solution to be employed is made up as follows:

Acid tartaric (Merck's reagent), gr. j.gm. .075
Alcohol, ʒ v .....cc. .20
Aqua destil., q. s., ʒ vj.....cc. .200
0.375 to a litre 1/200 of normal.

The alcohol is added to prevent the formation of fungus growth, but not in sufficient quantity to precipitate the albumin of the blood in any morbid condition.

Method of Employing the Instrument.—A drop of blood is obtained from the finger tip or lobe of the ear in the usual manner. The test tube is held horizontally, and its contained blood pipette fills automatically by capillary attraction when the sharpened end is touched to the blood-drop as it emerges from the wound. With an ordinary minim pipette, with a piece of rubber tubing over the free extremity into the test tube, wash this column of blood with distilled water up to the point 0 (zero), which is the first division from the bottom, holding the test-tube vertically. Close the opening in the bulb of the test tube with the thumb and invert the tube several times, to cause a thorough mixing of the blood and water. The *reagent pipette* is filled with the acid reagent, and the rubber tubing is fitted over the sharpened end of the blood pipette; by compressing the rubber bulb the acid solution is forced through the pipette into the test tube, the aperture in the glass bulb being closed before the pressure is relaxed to prevent the mixture of the acid solution and the blood. Having done this, the test tube is inverted several times while still being attached to the reagent pipette, care being exercised to keep the reagent pipette in a vertical position, to avoid a gravitation of the acid solution into the rubber bulb of the reagent pipette, thus preventing a chemical action upon the rubber nipple with the formation of a flocculent precipitate. The interval (Fig. 1, A) between the closed end of the tube and the first marking, 0, should be placed in the cleft of the spectroscope (Fig. 1, D) and observations be taken as to the existence of bands of oxyhemoglobin. Should these bands be present the careful addition of acid solution is necessary. As the bands become fainter and fainter we know that we are approaching the point of neutralization, and it becomes necessary to add the acid, one drop at a time; then invert the test tube and examine with the spectroscope as before. After performing several tests with this instrument it will not be necessary to apply the spectroscope so frequently to determine the point of neutralization, as the eye quickly learns to detect this characteristic change by the color of the blood mixture. When the bands of oxyhemoglobin suddenly disappear the observation is at an end. It is then only necessary to read the result from the scale on the test-tube, which is graduated in cubic centimeters and the equivalents expressed in milligrams of sodium hydrate to 100 cc. of blood.

As a uniform light is necessary for physiological experiments, artificial illumination should be selected as being always available; an open gas-light was employed in our experiments entirely, and the relation of the spectroscope to the source of illumination kept always constant.

The scale of equivalents computed from a basis of 15 milligrams of blood to 2 cc. of acid solution, 1/200 of the normal, is as follows:

cc. of reagent	Mgs. of NaOH to 100 cc. of blood
2.6	345
2.4	319
2.2	292
2.0	266
1.8	239
1.6	212
1.4	176
1.2	159
1.0	133
0.80	96
0.60	79
0.40	53
0.20	26.6

It is not customary to express the alkalinity of blood by the quantity of acid required for its neutralization, but in milligrams of an alkali, sodium hydrate being generally selected for the purpose. The calculation will be as follows: The equivalent weight of tartaric acid $\frac{H_2C_4H_4O_6}{2} = 149.64$. Tartaric acid being dibasic, one-half the number of molecules

are taken to make a normal solution which equals 75 grms. (approximately) to a liter; for the present solution there is taken 1/200 of the normal, which is equivalent to 375 mgs. to a liter. Sodium hydrate has 40 atoms to its molecule, which gives an equivalent of 40 grms. to a liter, therefore 1/200 of the normal tartaric acid solution is equal to 40/200 or 200 milligrams, and each cc. of acid solution will give an equivalent of 0.0002 mgs. of the alkali.

Every 100 cc. of blood will equal 266 mgs. of sodium hydrate.

The usual method of obtaining the results is to estimate by mathematical calculation the amount of alkali present in the blood; but this method has always proved itself to be both tedious and inconvenient, as it requires a decided tax on the memory to recall the various estimates as a working basis. In place of this rather intricate method, I would like to offer the following as its substitute: I have devised for this pur-

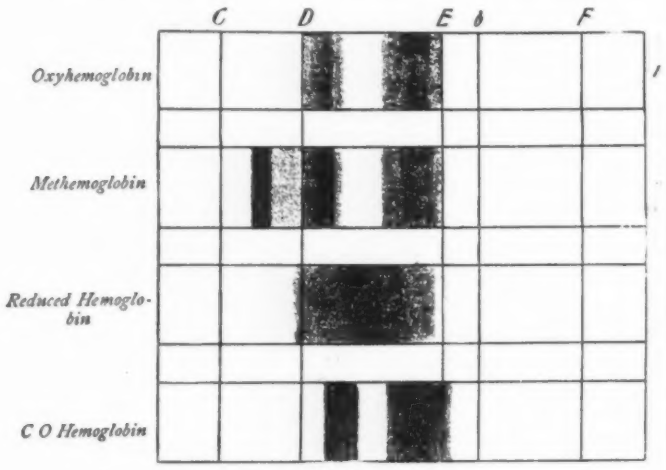


FIG. 2.

pose a scale of equivalents, in mgs. of sodium hydrate to 100 cc. of blood. Thus, should we employ 2.2 cc. of reagent, the alkalimeter tube would indicate an equivalent of 292 mgs. of sodium hydrate to 100 cc. of blood.

Very little can be added to the present knowledge of the alkalinity of the blood, beyond what has been learned from a study of a few cases. There is a more or less constant relation of the alkalinity of the blood to the color index, and our deductions are drawn principally from this discovery.

If this relation is borne out by the experiments of other investigators, we have at least a reliable basis upon which to make comparisons. The reason why the results obtained by different experimenters differ so widely is at once apparent, for as the disease progresses the color index changes with the varying relation between the number of erythrocytes and the hemoglobin. Even under physiological conditions marked variations of color index and alkalinity occur. The same pathological process in one case or in a series of cases, at various periods, gives diverse results; but should a definite relation between the color index and the alkalinity be established, we have a means by which the alkalinity can be studied even with the ever changing conditions of the plasma.

For sake of illustration a few cases are recorded to exhibit the relation between color index and the alkalinity in various morbid conditions. Many more cases were examined, but only those included, which were confirmed by clinical or laboratory methods, by surgical procedures and by post-mortem studies.

The alkalinity was included in the routine blood examinations made in Jefferson College Hospital by Dr. John Funke, resident pathologist, to whom I am indebted for the report of the 75 cases here presented; the table seems sufficiently interesting to merit a closer study of the relation of the alkalinity to physiological and pathological alterations of the blood.

From the seventy-five cases here tabulated it would be difficult or impossible to arrive at any definite or accurate conclusions, owing to the limited number of the cases under observation. In enteric fever a marked decrease in alkalinity is the rule. Thus, in 23 cases where a clinical diagnosis of enteric fever was made, it was discovered that in 20 of them the alkalinity of the blood was normal or below normal. As the tests were made at the time that the blood counts were asked for (regardless of the duration of the disease) it would be impossible to offer any explanation as to the exact period when the decreased alkalinity manifested itself.

A decrease in the alkalinity is likewise observed in tuberculous disease with glandular involvements, while with the existence of tuberculous meningitis or peritonitis the alkalinity shows an increase or falls late in the course of the affections.

In gastric ulcer there is a fall in the alkalinity below the color index, while gastric cancer shows an alkalinity above the color index. It may be here stated as a general fact that in all malignant conditions the alkalinity of the blood was found to be normal or else to have shown some increase in its alkalinity.

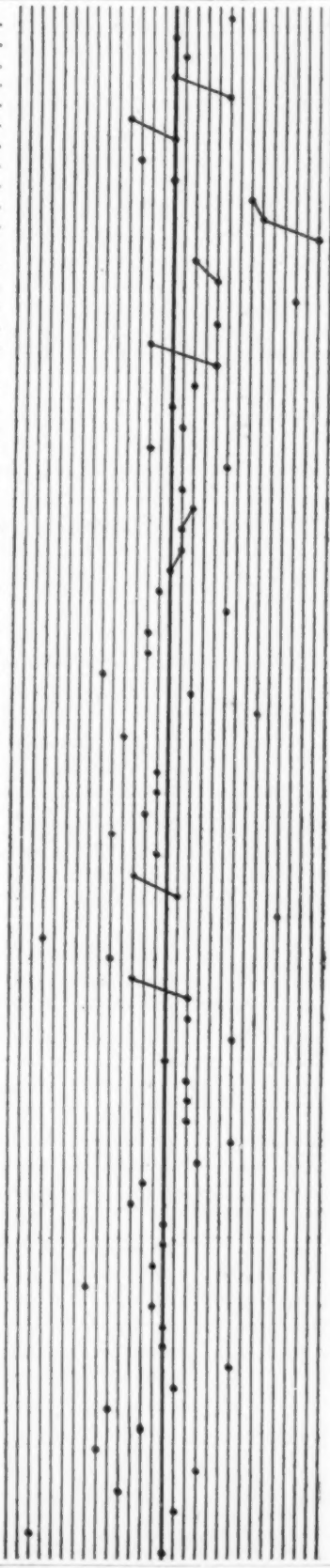
The anemias exhibited a decided fall in alkalinity, while observations upon chlorotic subjects showed the alkalinity to follow closely the normal color index. In the latter disease attempts were made to increase the alkalinity by the administration of excessive amount of sodium bicarbonate; and, while there was noted an increase in the erythrocytes and the existence of a turbid and alkaline urine, the hemoglobin failed to respond to the test, nor did the alkalinity rise above the normal; in other words, the previous existing relation between the alkalinity and the color index was still preserved.

In splenomedullary leukemia the alkalinity suffers a marked decrease, while in splenic anemia and also in cholemia there appears a rise in the degree of the alkalinity.

Catarrhal jaundice and amyloid liver both raise the alkalinity of the blood; cholelithiasis presented an evident decrease. In a case of abscess of the liver the fall in the alkalinity was most pronounced (123 mgs. to 100 cc. of blood).

Croupous pneumonia, phthisis, influenza, rheumatic fever, rubeola, appendicitis (acute and catarrhal), even in the pres-

TABLE.



Disease.	Erythrocytes.	Leukocytes.	Per ct. hemoglobin.	Color index.	Alkalinity.	Widal reaction — or ×	Per ct. urea.
Enteric fever	5,240,000	6,400	85	0.81	1.1		
" "	3,810,000	5,800	63	0.82	1.6	×	
Supposed enteric et chr. bron.	4,590,000	6,400	75	0.81	1.5		
Enteric on admission	4,220,000	12,800	70	0.82	1.65	—	
Thirty-five days later, temp. norm. 7th day	2,640,000	13,400	69	1.3	1.5	×	
Enteric on admission	4,570,000	16,400	88	0.96	2.3	—	
" later	5,300,000	9,500	80	0.75	1.5		
"	5,240,000	16,000	68	0.64	1.5	—	
"	4,460,000	9,400	76	0.85	1.7		
" Mrs. H.	3,700,000	4,000	65	0.87	1.0	×	
" three days later	3,350,000	3,800	54	0.80	0.8	×	
" five days later	3,660,000	3,800	54	0.72	0.1	×	
" Mrs. G.	4,710,000	7,800	74	0.78	1.3	×	
" five days later	4,040,000	8,000	80	0.99	1.5	×	
" Mrs. M.	3,200,000	7,200	69	1.1	0.9		
" Mrs. C.	3,460,000	7,400	70	0.88	1.3	×	
" Mrs. T.	4,980,000	15,900	70	0.70	1.6		
" six days later.	5,150,000	6,600	68	0.66	0.9	..	2
"	3,570,000	9,000	85	1.1	1.8	—	
"	3,910,000	19,400	86	1.1	2.0		
"	4,516,000	13,400	87	0.95	1.8	×	1.7
"	6,240,000	3,800	79	0.71	1.6	×	1.8
" et pneumonia	4,430,000	13,000	76	0.85	1.2	×	2.4
Chlorosis	4,540,000	9,600	55	0.65	1.2		
" } Treated with	2,950,000	8,400	20	0.34	0.35		
" three days later. } enormous doses	3,450,000	5,400	24	0.34	0.5		
" six days later. } of sodium	3,390,000	.....	24	0.35	0.6		
" ten days later } bicarbonate.	4,130,000	.....	25	0.31	0.6		
"	4,560,000	.....	27	0.29	0.6		
Splenomed. leukemia	2,880,000	127,000	38	0.65	0.8	..	2.2
Splenic anemia	4,010,000	3,600	65	0.81	1.8		
Catarrhal jaundice (coag. eight mins.)	4,450,000	11,400	66	0.74	1.6		
" "	4,970,000	10,000	75	0.75	2.1		
Cholelithiasis (coag. eight mins.)	4,770,000	5,800	59	0.61	1.0		
Abscess of liver	1,990,000	90,000	23	0.58	0.3		
Amyloid liver (digitalis poison)	5,230,000	9,700	64	0.61	1.6		
Croupous pneumonia	4,590,000	11,600	67	0.72	1.5		
" " (low temp., fatal)	4,170,000	25,800	60	0.71	1.5		
Pulmonary tuberculosis	4,800,000	10,400	68	0.70	1.55		
Rheumatic fever	4,620,000	.....	60	0.64	1.7		
Influenza	4,530,000	10,800	70	0.76	1.6		
Rubeola	4,370,000	19,000	46	0.52	1.3		
" fifth day	5,300,000	4,000	67	0.63	1.1		
Tertian malaria (morphinism)	3,240,000	4,400	49	0.75	0.5	(Hyoscin)	
" " (recent infection)	5,990,000	8,200	68	0.57	2.2		
Tubercular meningitis	3,900,000	8,200	67	0.98	2.35		
" peritonitis	5,010,000	8,200	68	0.67	1.6		
" twenty-four days later	4,410,000	8,100	68	0.77	1.3		
" peritonitis	3,260,000	13,200	21	0.32	0.4		
" adenitis	4,580,000	6,200	78	0.85	1.05		
" "	4,990,000	13,600	50	0.59	1.1		
" "	4,970,000	9,700	65	0.65	1.1		
Gastric ulcer	3,400,000	13,800	45	0.66	1.1		
" "	3,680,000	3,000	65	0.88	1.45		
" "	2,700,000	6,200	40	0.75	0.9		
" "	3,340,000	7,800	46	0.68	0.95		
Carcinoma ventriculi	4,300,000	6,000	40	0.46	1.1	..	1.8
" of breast	3,550,000	9,000	69	0.97	1.6		
Malignant disease of mediastinum	4,460,000	11,000	60	0.67	1.3		
Mal. dis. of liver, gall-bladder, or pancreas	4,190,000	12,800	55	0.66	1.3		
Sarcoma of thigh	5,230,000	5,950	75	0.72	1.5		
Lymphosarcoma of neck	5,370,000	19,200	76	0.67	2.0		
Carcinoma of breast	3,700,000	14,600	70	0.94	1.9		
Malignant disease of groin	5,310,000	7,200	80	0.75	1.5		
Diabetes mellitus	5,300,000	11,000	85	0.80	1.6		
Parenchymatous nephritis	2,360,000	5,200	33	0.69	0.7	..	0.5
Inflammatory condition of round ligament	3,950,000	14,200	67	0.84	1.5		
Renal calculus	5,470,000	7,600	78	0.71	1.9		
Appendicitis	5,450,000	22,200	90	0.82	1.8		
" catarrhal	5,270,000	8,600	80	0.76	2.1		
Cellulitis (leg)	3,500,000	22,000	25	0.55	0.8		
Appendicitis (fourth day of attack)	6,150,000	13,900	67	0.54	1.4		
Osteitis defor. et int. nephritis	4,950,000	5,800	70	0.71	1.3		
Angioneurotic edema et urticaria	6,350,000	10,400	84	0.66	2.45		
Normal	5,000,000	6,000	100	1.0	2.0		

To determine the relation of the alkalinity to the color index, multiply the color index by two (2); the difference between this result and the cubic centimeters of reagent, required to neutralize the blood, represents the alkalinity above or below the normal (color index.)

ence of a frank leukocytosis, showed an increased alkalinity, while investigations upon malarious subjects appear contradictory. In one case of chronic malaria, where the patient was addicted to the use of morphin, treatment was effected by the use of hyoscine hydrobromate, and the alkalinity was then found (133 mgs. NaOH to 100 cc. of blood) to be subnormal, while on the contrary a recent infection with tertian malaria presented an alkalinity above the normal (133 mgs. NaOH to 100 cc. of blood), the color index in the first being 0.75, in the second 0.57.

The relation that the alkalinity bears to the excretion of urea needs investigation.

Imperfect as have been these studies, they would tend to show that in those diseases which present a marked decrease in the alkalinity, they at the same time exhibit a marked increase in the quantity of urea eliminated or the amount of nitrogenous food is diminished.

JOHN COLLINS WARREN.[1]

By J. G. Mumford, M. D., *Boston.*

Unlike the history of literature or of politics, the history of medicine must be largely an account of men and the lives they led. Good literature produced in the eighteenth century is good literature in the twentieth century. The political acts and motives of our ancestors find their counterpart among us today, and the value of such former things is vital still; but the science of ancient times is no longer our science. With few exceptions that old knowledge is but as the babbling of children in our ears. We cannot take seriously most of the former medical wisdom; but we may honor the men and follow their lives and admit the debt we owe them; for out of their much talking and blind groupings there grew the scaffolding of that edifice which we are now building.

When we come down to the men of our grandfathers' generation these thoughts are not so obvious, because in them we begin to see ideas and motives and acts more modern. But much that they did well has now come to seem very feeble and erring—little worthy of serious study, though doubtless very important and urgent a hundred years ago.

However, the reading of that old medical literature is still very interesting. The acumen of the men, with the dim light they had, is remarkable indeed; their shrewd guessing, their frequent missing and infrequent hitting of the mark make a study of constant pleasure and charm for the student of those times. Yet an account of such hittings and such missings and guessings must form no part of this present writing. The task would be too intricate and lead us into technicalities too far afield. After all, it is the men themselves whom we must observe, and those few things which now and again they did greatly.

Among the men of that generation of our grandfathers few led more steadily laborious and useful lives than did John Collins Warren. He was born in Boston in 1778, on the first of August, the eldest son of that interesting John Warren[2] who served in the Revolution and founded the Harvard Medical School.

If ever there was a man blessed, or cursed, as you choose, with the New England conscience, it was John Collins Warren. His father wanted to keep him out of medicine, and he himself had no natural liking for it. We have it over his own signature that he was indolent and hated study; yet, once having put his hand to the plow, he never turned back, but devoted himself heart and soul, steadily, faithfully, without enthusiasm, to his profession, for more than fifty years. And he certainly had a very marked influence upon general practice in Boston, upon teaching at Harvard, upon surgery at the Massachusetts General Hospital, and upon his professional brethren in this country. Intellectually he was unlike any of the other early American surgeons, though in certain elements of training and experience he might be compared to Valentine Mott of New York. Like Mott, he was the son of a doctor, he was born and reared in an old, well established community, and in a medical atmosphere; he was thoroughly educated for his work and he spent his life in the midst of congenial surroundings, social and professional.

We probably know as much about him as we could know about any man of his temperament; for he had a steady appreciation of his own position in life and took copious biographical notes of his own career; those notes were elaborately edited by his brother soon after his death.

Inheriting a strong position from his distinguished father, he had a constant and proper pride in supporting it, and the combination of a sound understanding, wide culture, laborious industry and eager grasp of opportunity, together with the fortunate circumstance that for many years he met with little serious professional competition, secured for him in early life the unique position of surgical autocrat of New England.

His own biographical notes contain abundant material for a delightful memoir of his times, if only they had fallen into the hands of a Trevelyan or a Lang. Unfortunately, his editor was too much bent on eulogy for the popular success of the book. In spite of these drawbacks, we have a picture of a very important and very full career, and of a man familiar in his day to doctors throughout the land.

[1] Read before the Johns Hopkins Hospital Historical Club, February 9, 1903.

[2] The four generations of surgeons of this well known family are: John Warren, 1753-1815; John Collins Warren, 1778-1856; Jonathan Mason Warren, 1811-1867, and John Collins Warren, 1842, who is still active among us.

Warren's youth was passed in surroundings which seem very ancient to us now. His grandson and namesake has given us a charming sketch[3] of those old days, taking his material from his ancestor's own notes, which run:

"At the period when I left college and became an inhabitant of Boston it was thought necessary to undergo the operation of a barber half an hour every day. This consumed much time, besides the horrid consequences of carrying on one's head a quantity of curls, pomatum, flour, and the long cue or heavy club.

"The dress at that time was a colored coat with metal muttons, usually yellow; colored waistcoat, short breeches, buttoning at the knees; long boots with white tops, and when riding on horseback a pair of leather breeches instead of pantaloons, of drab cloth.

"These yellow breeches were daily cleaned with yellow clay, which required that the coats should never be brought in contact with them. Then a short ruffle at the breast and about the wrists, a white cravat, filled out with what was called a pudding, the use of which, from the effect of habit, could not be dispensed with for some years.

"Cocked hats were very much worn at the time, but not by the young.

"Gentlemen of a certain age wore wigs, which were sent to the barbers once a week to be fresh dressed, so that on Saturday night we saw the barber's boys carrying home immense bundles of wig-boxes as a preparation for going to church on Sunday.

"Physicians who had much business in those days rode on horseback. Riding in a chaise was very rare, and in a four-wheeled carriage still more so. My father rode on horseback till a few years before his death.

"Dr. Lloyd generally drove a very fine horse, and Drs. Jarvis and Whipple were famous for beautiful saddle horses and the elegance with which they rode.

"Large parties opened at 7 or 8 o'clock in the evening, and were much more formal than at present. A friend of mine told me that he saw me dance a minuet in 1796 or thereabouts, and that this was the last time he had witnessed this dance in Boston.

"Persons of a certain age were treated with a degree of deference now wholly disused. In fact, one of the great traits of the manners of the present time is the manner with which young persons are accustomed to treat persons older than themselves.

"Gentlemen's dinner parties began early and ended late. The great care on the part of the host was to present to the guests as much ordinary wine as they could be made to drink, and then to bring forward in succession a variety of old wines, each having a character a little better than that which preceded. All of these had some remarkable history connected with them, the details of which constituted an important part of social discussions.

"On the whole, the dinner parties of those days must be looked on with disgust; for not only was the quantity drunk sufficient to make irreparable inroads on the physical organization, but this indulgence led to coarse extravagance and thought, and the conversation at a dinner party, if taken down by a stenographer and presented to the party on the morning following, would have filled them with shame and regret."

Warren was intended by his father for a mercantile life, but the suitable opening not immediately presenting itself after he left college, he passed a couple of years at French and the *pretended* study of medicine, as he himself says. Then he went to Europe and settled down to serious work—that was in 1799 when he was 21 years old. His course there was much like that of many other young American students of the time. London claimed him first, where he became a pupil of William Cooper, and later of William Cooper's nephew, Astley Cooper. The elder was an able, conservative surgeon, testy and ignorant of all things beyond his little island—he was the antipodes of his brilliant, generous, popular nephew. Warren secured a dresser's position at Guy's Hospital—it was merely a matter of money down—and served at such work and dissecting for something more than a year. Then he went to Edinborough for a year, where he received his medical degree, and then for a final year to Paris. In the two latter places he studied hard—going in for chemistry, general medicine and midwifery, as well as anatomy and surgery. He lived in Paris with Dubois, Napoleon's distinguished surgeon, and studied chemistry with Vaquelin and Fourcrois; anatomy with Ribes, Sabbatier, Chaussier, Cuvier and Dupuytren; medicine with Corvisart, and botany with Desfontaines. That was a brilliant gathering for the edifying of a young gentleman from Boston. He says that the French students were green from the Revolution, for the most part a rude and vulgar set of people, who made him much trouble first and last.

In the autumn of 1802, Warren came home, by the way of England, and on arriving in Boston, found his father in very poor health. In order to relieve him he immediately assumed a great part of his practice. It is said that at this time the elder Warren had a larger private practice in Boston, than any other physician has carried before or since. At any rate the son found himself almost swamped by these new duties, and tells in his diary that frequently during the year he would make fifty professional visits a day. Allowing for a working day sixteen hours, this would give him about twenty minutes to each patient, not counting the time consumed in travelling; and, as much of his work was midwifery, we must think of him as a young man with an extraordinary burden to bear. It is recorded of both the Warrens that they acquired a very great facility in dealing with patients and a remarkable intuitive skill in the diagnoses of the day. Their visits were purely business-like. They would take up the case at once, wasting no time in gossip, and the required duty being completed, would promptly withdraw.

This very sensible method, so unlike that usually followed

[3] Reminiscences of An Old New England Surgeon (Maryland Medical Journal, 1901, vol. 44, p. 45), by John Collins Warren, M. D., F. R. C. S.

by their colleagues, gained them the respect of their patients and saved hours of valuable time, daily.

The fortunate young Warren found other advantages to his hand—not the least among them an opportunity for teaching. His father was finding that work and the ride to the Harvard school in Cambridge were almost more than he could bear, so the son was set to work to relieve him. The Harvard school was still in its infancy. Its distance from Boston made it difficult of access for students living in the city, and there was, of course, an absolute lack of clinical material in Cambridge. At about the time of Warren's coming home, James Jackson also appeared upon the scene, and we find the names of the two men associated thereafter for more than fifty years. They and their contemporaries were joined by the elder Warren, and by Dexter, Professor of Chemistry, in the effort to transfer the school to Boston, and after years of rather bitter conflict with Waterhouse, Professor of Practice, they succeeded in effecting the change in 1810.

Those years between 1802 and 1810, were important years to Warren in many ways. He married a wife, to begin with, in 1803, a daughter of Jonathan Mason, and began the rearing of his many children. He was active in all sorts of literary, social and scientific enterprises. With John Lowell, J. Q. Adams, Kirkland, Quincy, Jackson, William Emerson and others, he started a Natural Philosophy Society; with Gardner, Emerson, W. S. Shaw, Buckminster, Tuckerman, Jackson and others he established the Anthology periodical and the society which grew into the Boston Atheneum; with Jackson, Dixwell, Coffin, Bullard and Howard, he formed a Society for Medical Improvement. In 1806 he was made Adjunct to his father in the chair of Anatomy and Surgery at Harvard, and he succeeded to the full professorship, upon his father's death, in 1815.

Warren's name will always be associated with two important facts. One was the founding of the Massachusetts General Hospital; the other was the introduction of ether anæsthesia. These two events were separated by an interval of twenty-five years, but around them both are grouped nearly all that is conspicuous in Boston medicine during the first fifty years of the last century. There were other men of course, concerned with both events—some of them concerned more intimately than was Warren; but Warren was part of both and for such distinction is known to us.

Before the establishment of the hospital, his constant occupation as a teacher and general practitioner led him into lines of research less strictly surgical than what we know of his work in his later years. In 1809, while still comparatively fresh from European teachers, he published a valuable paper on organic diseases of the heart, a subject, which until then was little understood in this country; and in 1811, together with Jackson, Gorham, Jacob Bigelow and Channing, he assisted in founding the "New England Journal of Medicine and Surgery." This publication was ably edited and in 1828 was united with another, under the title "The Boston Medical and Surgical Journal," which flourishes today.

As a writer, Warren was lucid and strong. He had a great many things to say, and he said them well. His belief was that, as yet, the profession in America was too young and inexperienced for original work of moment. He recognized the inadequate training of the great mass of his fellows, and his conviction was that their first need was to acquire and absorb the learning of the old world. This belief he preached with pen and by word of mouth. He was one of the first to see the true functions of a medical school, and he followed other wise men in insisting upon the establishment of hospitals. His lectures were carefully prepared, and systematically delivered. He was clear and instructive without being eloquent. He lacked the charm and magnetism of his father and uncle, Gen. Jos. Warren, in public speaking, but he was more learned than they and ably carried on the work which his father had well begun.

The Massachusetts General Hospital was slow in getting started. So long ago as 1810, Jackson and Warren organized the movement for its foundation—a movement which had been in the air for many years—if, indeed, it may not be regarded as the direct outgrowth of that great military hospital in Boston, over which old John Warren went to preside in 1777. When that old institution disappeared after the war, the elder Warren missed it sadly, and in all his later life essayed to promote a proper substitute.

At last, on the 10th of August, 1810, these various desires of wise men in Boston found voice in a circular letter signed by James Jackson and John C. Warren, in which they asked their fellow townsmen for subscriptions to a "hospital for the reception of lunatics and other sick persons." Promptly the good work was taken up by prominent men of the laity—James Bowdoin and fifty-five others—citizens of Massachusetts—incorporators under the title "The Massachusetts General Hospital," and the enterprise progressed slowly and substantially, until finally in September, 1821, the first patient was received. Warren was then forty-three years old, and his father had been six years dead, without seeing the long awaited hospital an accomplished fact.

It was to the two men, Jackson and Warren, that we owe the Massachusetts General Hospital, and to this day their names, and the name of Bigelow, are the ones most closely associated with it. From the outset, it was a general hospital, where acute diseases were received, though from the beginning its wards for the insane were removed to a distance, located in Somerville, and named after John McLean, who had contributed towards the foundation, sums amounting to about $120,000.

Until the establishment of the general hospital, there was little opportunity in Boston for study and experimentation on surgical lines, but with the hospital's advent, Warren's career expanded in a fashion unknown even to his father, and in his own careful, methodical and painstaking manner, he proceeded to organize a routine for the surgical staff, in a fashion which has to this day left its impress upon the practice of Boston. Warren was very able, but, unlike many other able men, he was a man of detail. His whole life seems to have been schematic and his hospital practice was made to

correspond with the rest of his life. We have seen that he was keenly alive to his own dignity and position, and this characteristic, which sometimes was found irritating to his equals in the outside world, was made to serve a very useful purpose within the hospital walls. From the outset, his department was conducted on lines of almost military discipline. His colleagues were formally addressed and consulted, and the nicest punctilio was observed between them. His juniors on the staff were required to hold towards him and the other senior surgeons a proper distance and respect; the house staff, as we should call the young graduates who act as assistants, were enrolled as "house pupils," were addressed as "Mister" and were not permitted to assume the title of their doctor's degree until the end of their year of service. Their duties were strictly those of the humblest of assistants, they were given no responsibility beyond the very slightest, and their labors consisted largely in the careful writing at dictation, of voluminous records, and in aiding in the dressing of wounds. To some extent, too, they did assist at operations, though often this work was done by one of the Surgeon's own colleagues. In their turn, the nurses and the servants of the hospital were relegated to the very humblest of positions. The result of all this was a most admirable machine, which once established, ran of itself; and in some measure still runs on, much as it was set going eighty years ago.

The position of a surgical teacher, who is also at the head of a hospital, on whom students wait for their instruction, and young doctors for their orders, is almost unique in civil life. The college president or the great merchant is less of an autocrat in his own field. The colonel of a regiment or the captain of a man-of-war alone surpasses him; and we must believe that Warren, with his temperament and opportunities, developed to the full the possibilities of the situation.

He was a very able surgeon of the painstaking type. In those days all operations, even the most inconsiderable from our point of view, were serious matters. Without antiseptics, there was always a probability of serious inflammation following, and without ether there was always intolerable pain. So the knife was used sparingly and of dire necessity only, and he was thought the skillful surgeon who could avoid it longest. Warren prepared with the greatest care for each operation. He read up his authorities, he consulted his notes, he studied his case and he practiced on the dead subject. By such practice he became rarely facile, but he never presumed on his facility. His work on the living was done methodically and with minute pains, to avoid hæmorrhage or damage to structure. His work was thoroughly done and it was well done. In all the minutiæ of dressings, bandaging and apparatus he was a past master. He held that, so far as possible, wounds should be closed without stitches, and his dressings were works of art. Indeed, in such work he set the pace, and the rare beauty and method of bandaging and apparatus in Boston hospitals are conspicuous to this day.

In one respect, his admiring brother and biographer does him an injustice, for he tells of his brilliancy in diagnosis and how he would form his opinion at a glance. For the great majority of common lesions this was doubtless true, but it is hard to believe that a man of Warren's careful and accurate mind would give way to the temptation of what we call "snap diagnosis," especially in those ancient days, when instruments of precision were lacking and the science of pathology was just struggling into life. We have copious notes of his clinical remarks, made at the meetings of the Boston Society for Medical Improvement, and such remarks leave us with a feeling that he came to his conclusions only after careful thought and the exhaustive comparing of conditions.

About the time of the hospital's founding, Warren was at the height of his career. He had always been rather delicate in health, and it had been his constant care to guard a chronic dyspepsia, to have his body in condition and to keep the machine in good working order, as we say. Yet he was a very laborious man. It was his habit to rise early on winter mornings and breakfast by candle light; then he went out and made professional visits until one o'clock, when he dined, giving himself about ten minutes for that function; he saw patients until two, when he lay down for an hour's rest. In the latter half of the afternoon, he made further visits, supped at seven and spent his evenings until two o'clock in the morning, at his books and in writing. On hospital and lecture days his labors were still further prolonged. It was not an easy self indulgent life.

With all his care and method, Warren was not a timid operator. His amputations were bold and brilliant; he removed cataracts with great success; he taught and practiced the operation for strangulated hernia—the first surgeon in this country to do so, and against strong professional opinion here; he introduced the operation for aneurism according to Hunter's method. His excisions of bones for tumor, especially of the jaw, became famous and are classics—for are they not recorded in volumes of the "Boston Medical and Surgical Journal?" In 1837, when fifty nine years old, he published his *magnum opus*, "Surgical Observations on Tumors," a thick octavo with plates—a great collection of cases and remarks, interesting and instructive today. But all this gives only a very faint idea of his ceaseless literary activity. He was always writing; reports, memoirs, essays, lectures poured from his pen. It was a fluent pen, and had behind it a brain stored with keen thoughts and abundant information.

His extra professional interests were multitudinous. Few men have felt so constantly the burthen of their responsibilities to the community. He was like an officer on dress parade, or like a careful father in the presence of his young children—always punctilious about appearances. He felt it must not be said that a man of his eminence or importance ever yet set a bad example or appeared to disadvantage. So he was forward in good works and had a hand in whatever was going. He was dogmatic and final in his decisions and explicit with his advice. In matters surgical, his word was law for many years. It was not until his old age, and with

the advent upon the scene of another strong, young and aggressive man, that his power began to wane.

With his own advancing years, he also saw his son, J. Mason Warren beginning to gain the public confidence, and so he found time to devote himself more to work outside of his profession. He was always greatly interested in comparative anatomy and paleontology. He was able to secure, among other trophies, the most perfect skeleton of the mastodon which exists—the monster is still preserved in the old building on Chestnut Street which has been known for sixty years as the Warren Museum. All through his life he devoted himself, like Hunter and Cooper before him, to the collection of anatomical specimens. This collection, together with the treasures of the Medical Improvement Society, passed years ago to the Harvard Medical School and formed the nucleus of the fine "Warren Museum" of that institution. He became a convert to total abstinence in middle life, and was for many years president of the Boston Society for the Promotion of Temperance in the use of alcohol. He was also president of the Natural History Society of Boston and was an active member of the Agricultural Society. An interesting organization for the promotion of scientific and literary pursuits, was founded by him; it included in its membership many of the most eminent and scholarly men of Boston and was at first known as the "Warren Club." Under the name of "The Thursday Evening Club" it flourishes to this day, with a history of unbroken excellence and interest, and at the present writing is presided over by the grandson of its founder.

Warren's acquaintance with the profession in this country and in Europe was extensive. He was more given to travelling than was the wont of Bostonians of his day, and he made an extended trip to Europe, when in middle life; renewing old acquaintance, seeing society—indeed he was present in London with his family at the time of Queen Victoria's coronation—and visiting the homes of science at the fountain head.

He was prominent in the establishment of the American Medical Association, of which the primary purpose was the elevation of medicine in practice and in teaching, and he was one of its early presidents. Of all these things we have the story admirably told by himself in his published biography.

Then there was that other great event with which his name is most conspicuously connected; the first public use in surgery of ether anæsthesia. That was in October, 1846, when he was approaching his seventieth year; an age which we are not wont to associate with great and daring progress in affairs. It is needless here to enter upon that most interesting and confused chapter of American surgery. Suffice it to admit as Jacob Bigelow admitted years afterwards, that to Warren belongs the credit, in his old age, of allowing his name and position to stand sponsor for this courageous and revolutionary experiment. Warren himself was too near the end of his career to benefit greatly by anæsthesia in surgery; but in some measure he saw its significance, and wrote about it and championed its uses always.

The old man lived on until 1856. Fifteen years before his death his wife died, leaving him with six grown children, and two years later he married a daughter of Governor Thomas Lindall Winthrop, who also died before him.

He kept busy almost to the end of his life, especially with his writing. His last surgical paper was published in May, 1855, just a year before his death, which closed a brief and painful illness.

His real work had been done long since, however. It is not a life which lends itself readily to eulogy. It was not full of striking events and dramatic incidents. Except for the ether business no event stands out conspicuously; and in that he but lent his name, as, indeed, but for him some other might have done. But it was his long and useful career that made him eminent; his services in helping to found a great hospital, his establishment of sound surgical methods, his correct and methodical teaching, his faithful searching out of the truth, his insistence upon drill, his contempt for the brilliant superficial. All these things were very important, and among us helped to set a new standard, up to which we have been growing ever since. He was indeed a man whose work our community could ill have spared; and though he was succeeded by the meteoric Henry J. Bigelow, the younger man would have found for his endeavors a very different field, had it not been so carefully and faithfully tilled through toilsome years by Warren.

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SERIES I-II-III.

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THE ETIOLOGY OF SEROFIBRINOUS PLEURISY WITH REFERENCE TO CYTOLOGICAL DIAGNOSIS.

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(From the Medical Clinic of Professor Osler.)

The etiology of the so-called "idiopathic" pleurisy with effusion in apparently healthy individuals has been a vexed question for over a half century. With the growth of our knowledge of bacteria as causal agents in disease, the early explanation that the inflammation was due to cold has seemed inadequate, and an attempt has been made to place the disease in the category of acute infections. From early suggestions by Pidoux, Bernutz, and Villemin the idea that many of the cases were of tuberculous origin has steadily gained ground in France, until in 1884 we find Landouzy taking the extreme position that "98 fois sur 100 les epanchements pleuraux; les pleuresies pretendues a frigore sont fonction de tuberculose apparent ou cachee" and to this view his countrymen seem to subscribe. For the Anglo-Saxon mind, however, it has seemed difficult to recognize the possibility of a tuberculous origin in an effusion coming on after mere exposure to cold, in an apparently otherwise sound, healthy, robust man, even more difficult than to realize that a small hemoptysis in a similarly apparently healthy person is a sign of tuberculous infection. As a result the French view has met with much opposition in England and in this country, the general view of the opposition being perhaps well expressed in the words of Prudden, who says in summarizing his investigations of effusions that "results seem to justify belief in the comparatively frequent occurrence of simple exudative pleurisy with serofibrinous exudate which is not tuberculous and not demonstrably associated with bacteria of any kind." Those who have chosen the middle ground have found that it slopes toward the Landouzy view and allows of but little lingering by the way.

The question, then, of the etiology of these pleurisies *a frigore* cannot be said to be absolutely settled. It has been attacked from almost every possible side; the method varying somewhat with the prevailing fashion in medicine at the time, but has proved a question that does not lend itself to easy solution. There is no *a priori* reason why the pleura should not be the seat of primary tuberculous infection. Indeed, when one notes at post mortem how carbon pigment and other foreign particles are apparently hurried by the lymphatic stream of the lung toward the pleura, there often to be lodged in the subpleural connective tissue, it would seem as if primary pleural might be even more frequent than primary pulmonary tuberculosis. The opportunity of directly determining the condition of the pleura in the acute stage of an uncomplicated pleurisy at post-mortem examination is rarely obtained, as a fatality at this period of the disease is an extremely infrequent event. These rare opportunities become of value statistically only if they fall into the hands of pathologists who perform the necropsy with the possibility of tubercles being present, well in mind. Medical literature seems peculiarly free from reports of such cases. The most satisfactory and at the same time the most startling statistics are those of Kelsch and Vaillard, who in sixteen fatal cases of acute pleurisy found tubercles on the pleura of sixteen. Of these sixteen cases, seven were serofibrinous, two hemorrhagic, four were first serous becoming purulent before death, and three were purulent when first seen. Of 147 cases of pleurisy in post-mortem cases, collected from literature by them, they found mention of pleural tuberculosis in 113, in 9 tuberculosis of other organs is mentioned, but no reference made to the condition of the pleuræ, and in only 25 is examination of the pleura for tuberculosis mentioned as negative. Few of these cases, however, fell in the category of acute uncomplicated pleurisies. Osler in analyzing the Johns Hopkins Hospital post-mortem records for his Shattuck Lecture in 1893, found of 101 cases 32 per cent were tuberculous; but of these eight were purulent and associated with pneumothorax, eleven were associated with general acute miliary tuberculosis, and thirteen only were of the chronic serofibrinous form. This last group only might be considered as belonging to the primary pleural affections. Barr reports definite tuberculous findings in the pleura of three cases of acute pleurisy dying within a few months from the onset of the disease, the earliest three months after onset.

Failing in the matter of large statistics on the direct post-mortem determination of the cause of the disease, there are open two avenues of investigation: the rather direct method of the examination of the exudate, and the indirect method of following the subsequent history of the patient. Along the latter line we have several carefully compiled sets of statistics, which show in general that pleurisy patients are very apt in short time from the onset of their disease to show signs of tuberculosis in other organs. Thus Fiedler reports that of 112 cases at the end of 2 years from the onset of the pleurisy only 21 were well. Of the others, 17 had died of tuberculosis while in the hospital, 8 had died shortly after leaving the hospital, and 66 gave definite signs of tuberculosis. Barr, in 74 cases, finds that 32 died within five years from the onset of the original disease, their aver-

age length of life being two and a half years. Of these 32, 21 died of tuberculous infections; seventeen of the cases could not be traced. Lemoine reports an apparent cure in but five of twenty-eight cases; of the others, one had tuberculosis at the time of his pleurisy, seven showed doubtful signs, and fifteen became definitely tuberculous later. Ricochon reports that of twenty-one cases, only ten were alive after a lapse of three years and they were all tuberculous. Bowditch reports in 1889 that of thirty patients who had acute pleurisy in the decade 1849-59, thirteen had died of phthisis; of nineteen in the decade 1859-69, nine had died; and of forty-one in the decade of 1869-79, ten had died; giving an average of 38 per cent of the 90 cases that subsequently died of tuberculosis. These cases are classed together without reference to the nature of the original disease, that is as to whether it was a "dry" pleurisy or pleurisy with effusion. Cabot's series of 152 cases, recently published, shows a decidedly smaller number of patients who became tuberculous subsequent to their pleurisy. Within five years but ten were affected, in the next five years eight more; in the next five years four more; and in the sixteenth year one more—a total of twenty-three, or 15 per cent. Twenty-one patients were living and sound between fifteen and twenty years after their attack; twenty-three between ten and fifteen years; and thirty-six between five and ten years, giving a total of eighty patients well five years or more after their pleurisy. Thirty-seven were alive within the first five years after the attack and fourteen had died of some illness entirely unconnected with pleural disease. Ninety per cent of the cases were well two years after the pleurisy.

While these statistics all point in one direction, they are in a sense not entirely satisfactory. One might take the ground that the original pleurisy, of whatever etiology, had, by its sequelæ of adhesions, thickened pleura, contracted chest, atelectatic portions of lung, etc., merely left the lung in a more receptive condition for infection by the omnipresent tubercle bacillus. On the other hand, when one sees post mortem how many healed foci of tuberculosis in the lungs may be demonstrated by careful search, he is not convinced that the thirty to sixty per cent of pleurisy cases that do not subsequently develop tuberculosis have not been able to withstand and take care of what was originally a tuberculous affection.

The aspirated exudate in pleurisies has been repeatedly studied bacteriologically by cover-slip, culture, animal inoculation, and serum reaction, and quite recently cytological examination has come into prominence. The cover-slip and cultural examinations give results that are capable of several interpretations. They led to the early recognition of what may be termed the bacteriological law of effusions, that bacteria are easily demonstrable in purulent but not in serous effusions, or conversely, effusions in which bacteria are easily demonstrable on cover-slip and in culture are purulent or tend to become so. Thus the large group of serous effusions has come to be regarded as sterile effusions. In these large effusions careful and tedious examination of sediment obtained by centrifugalizing relatively large quantities of fluid generally proves unavailing. If in some, cocci or other of the ordinary pyogenic group are found in cover-slip, they are generally not demonstrable in culture, an evidence of the bactericidal effect of the serum. The tubercle bacillus is almost never found on cover-slip in these serous effusions (though usually quite easily demonstrable in cases of pyothorax and pyopneumothorax secondary to pulmonary tuberculosis, and probably there to be considered as derived from some communicating pulmonary cavity). We find unanimous agreement on this point among all who have investigated effusions, among whom may be cited Ehrlich, Fraenkel, Wolff, Netter, Prudden, Osler, Dejerine, Renvers.

This group of sterile effusions then has been held by some to indicate that the whole process is a sterile process, leading to conclusions regarding "idiopathic" pleurisies similar to that of Prudden quoted earlier. On the other hand, in the French school, it is held to indicate that they are tuberculous in origin, the failure to find the bacilli being due in part to the fact that the bacilli are not easily dislodged from the pleura, perhaps due to the false membrane formed; and in part that if dislodged they are so few in number that in an effusion of one or two liters they offer "a needle in the haystack" problem as far as cover-slip preparations are concerned.

Tubercle bacilli may, however, be demonstrated in many of these effusions by the method of animal inoculation, and this method emphasizes the point last mentioned, as success is only to be obtained by inoculating relatively large amounts of the fluid. The method was first employed in 1884 by Chauffard and Gombault, who in twenty cases got ten deaths of animals from tuberculosis. Netter in twenty cases found eight positive; Widal and Ravaut, five out of seven; Eichhorst fifteen out of 23 or 65.2 per cent of cases; Le Damany forty-seven positive cases in fifty-five. Of the eight negative results four were clinically tuberculous. These results need no comment.

Some effort has been made to obtain agglutination of tubercle bacilli by the serum in effusions, but as yet the method has not proved of easy application or of much practical value. Debove and Renault have, however, obtained the tuberculin febrile reaction in tuberculous patients by injecting subcutaneously filtered pleural effusions; and conversely, Netter quotes statistics published by the Prussian government, in which Koch's tuberculin gave a positive reaction in thirteen out of fifteen patients suffering with pleural effusion. These statistics would seem to show that by careful laboratory work and by use of animals it is possible to prove the greater number of the so-called "sterile" effusions to be really tuberculous.

There has recently come in vogue a simpler method of examination by which it is hoped one will be able to differentiate this large primary group of cases with its bad prognosis from a much smaller group, probably usually due to a

small pneumonic patch, in which the exudate early may have contained a few of the pyogenic organisms which were soon killed through the bactericidal power of the exudate, cases which bear a much more hopeful prognosis. The method is that of the examination of the cellular content of the effusion, introduced by the work of Widal and Ravaut, published in 1900, and of Wolff published in 1901.

Widal and Ravaut divide effusions into three groups: the acute septic; the tuberculous (idiopathic), and the mechanical (cardiac and nephritic). The sediment of the first group, the septic cases, they find to be made up almost exclusively of polymorphonuclear cells, especially in streptococcus cases, with a few small mononuclears, a few endothelial cells, and red blood cells in cases due to the pneumococcus. Typhoid pleurisies tend to be hemorrhagic, but as to leucocytes, agree with the other septic cases. Of idiopathic pleurisies, they studied 56 cases, the effusion in all being taken as late as the ninth day from the onset of the disease or later. In these cases they find agreement in the fact that the white cells of the sediment are almost exclusively small mononuclear cells. In addition, are found many red-blood corpuscles, a few large mononuclear cells, a few large endothelial cells (apparently derived from the pleura), and only rarely a polymorphonuclear cell, except in two cases aspirated on the ninth day, in one of which there was about one polymorphonuclear cell to nine small mononuclears. They consider it of importance also that the endothelial cells are few in number and always isolated. Seventeen cases showed no endothelial cells. Animal inoculations were made in a number of these cases and at the time of their report, of seven of the experiment animals that had died, five showed tuberculous lesions. In the mechanical pleurisies, cells are fewer than in the two other groups. The characteristic cell is the endothelial cell from the pleura, and what they consider as pathognomonic are "placards" of two, three, or several endothelial cells without visible cell outlines.

Wolff's results agree in the main points with those of Widal and Ravaut. He, however, was successful in obtaining effusions from somewhat earlier cases, and added to the findings above given. He finds that very early in the disease the exudate in tuberculous cases is almost exclusively polymorphonuclear, and concludes that at first it is entirely so. The small mononuclear, however, soon becomes evident and at the third day may make up one-third of the cellular exudate, gradually increasing in proportion from then on until eventually the whole exudate may be made up of mononuclears. He concludes in general that the cellular method of diagnosis is more exact than the cultural, and that an exudate of one-half or more of small mononuclear cells speaks for the tuberculous nature of the process.

Gulland, without giving the details of his work, reports that his examination of exudates extending over several years, confirms the work just quoted. Although he has not had any cases as early as the third day, in his earlier cases he finds a minor percentage of polymorphonuclears. Among others, Barjon and Cade, Dopter and Tanton, and Courmont and Arloing have published confirmatory results. This method has been applied to other effusions and collections of pus in the body with somewhat similar results. Tuffier and Milian have found small mononuclear cells predominating in tuberculous peritonitis and hydrocele of tuberculous origin; Achard and Loeper in peritonitis, pleurisy, meningitis, and joint tuberculosis. Tissot claims that the pus of tuberculous abscesses contains a higher percentage of small mononuclears than that of those due to the common pyogenic bacteria, while Milian determines the same for the pyuria and hæmaturia of renal tuberculosis. Widal, Sicard, and Ravaut have shown the same for the meninges. These with other similar findings have led to the cytological doctrine that the small mononuclear leucocyte is the surest index to tuberculosis.

During the winter of 1901-02 a number of pleural exudations, in particular those from "idiopathic" pleurisies in patients in the service of Dr. Osler at Johns Hopkins Hospital, were studied as to their cellular content. While this number was not large enough to entitle one to draw any sweeping conclusions, yet the results obtained would seem to point to the fact that the cellular constitution of an exudate may be of some diagnostic value. My results are in general confirmatory of those already published. In the primary pleurisies the exudates were usually of large volume and of fairly high specific gravity, the limits being 1.014 and 1.022 with an average of about 1.018. They were about evenly divided between a slightly turbid greenish-yellow fluid and one with a decided blood tinge. The albumen content was high and the reaction slightly alkaline to litmus but never to phenolphthalin. Cellular sediment was abundant. I find there is a very definite formula to the cellular exudate; and that the time at which the exudate is taken is a marked factor in determining the particular proportion of cells. My earliest cases were taken at about the end of the first week if I may except one case, a colored boy whose history as given is doubtful. In these cases, three in number, the polymorphonuclears form from 15 to 12 per cent of the exudate, the other cells being almost entirely small mononuclears, with a few cells indistinguishable from the large mononuclears of the circulation. During the second week, as shown in four cases, there is a fall in the number of polymorphonuclears (relatively and probably absolutely also), the limits being from 5 to 3 per cent. After this time, according to six cases studied, it is often impossible to find a single polymorphonuclear on a slide, the exudate being almost entirely mononuclear. There is always to be found a fairly large number of red-blood corpuscles and a few scattered and isolated epithelial cells.

Two cases on the seventh day formed an exception to this rule, showing an exudate composed of almost equal numbers of polymorphonuclears and small mononuclears. One of these effusions was a terminal event in a chronic cardiac case who gave a history of onset with pain in the side, cough, and bloody sputum. The exudate was markedly hemorrhagic. While this cellular formula comes within Wolff's

tuberculous limit, it also agrees almost exactly with the formula Barjon and Cade have found in effusions associated with infarct of the lung, which seems a probability in this case. Unfortunately no post mortem was obtained.

The other case was in a man, who a week before admission had, after exposure, been taken with a chill, pain in the side, and cough, followed in a couple of days by bloody sputum. At the time of admission he had no fever and no pulmonary signs except the flatness due to the effusion. The effusion was small in amount and showed forty-five polymorphonuclears to fifty-five small mononuclears, while almost a half of the sediment was made up of large epithelioid cells. There seemed a probability in this case also that the exudate was secondary to some slight pulmonary involvement.

A third exception was the exudate in the colored boy which on the third day, unless his history was misleading, which seems probable, showed 100 per cent small mononuclears.

In none of the exudates was the tubercle bacillus obtained on cover-slips. Cultures were made from a fair proportion and were uniformly negative.

In two cases of pyopneumothorax, secondary to tuberculosis of the lung, the exudate showed a large percentage of polymorphonuclear cells, probably over 80 per cent, although it was difficult to determine this exactly because of the marked disintegration of the cells. In both, the tubercle bacillus was found in cover-slip. In a single case of metapneumonic exudate, in which the pneumococcus was demonstrated on cover-slip, the exudate consisted of about 96 per cent of polymorphonuclears to 4 per cent of small mononuclears. The exudate was purulent. In three mechanical exudates or transudates the specific gravity of the fluid varied between 1.005 and 1.015. The cellular sediment was much smaller in amount. Epithelioid cells formed quite a large percentage, existing singly and in the "placards" which Widal and Ravaut consider so pathognomonic. The other cells were mononuclears 96 to 99 per cent, and polymorphonuclears 4 to 1 per cent, with a relatively large number of red-blood corpuscles. Thus, except for the more numerous epithelioid cells and the "placards" a slide made from a hydrothorax sediment would be very difficult to diagnose from a slide made from the sediment of an "idiopathic" pleurisy. This is a diagnosis, however, for which one would seldom be obliged to resort to the microscope. Widal and Ravaut explain the "placards" as indicating a transudate and not an exudate; the slow mechanical filtration of serum through the pleura causing a loosening of the epithelium which falls off or is rubbed off by respiratory movements. The absence of "placards" in inflammatory cases Ravaut attributes to the formation of a false membrane on the pleural surface preventing marked desquamation or holding the desquamated cells in its meshwork of fibrin.

Wolff in his first paper was inclined to consider the pleuritic effusion a transudate and not an exudate chiefly because of the supposed nonmotility of the lymphocyte. Following the demonstration by Hirschfeld of the motility of the lymphocyte and his own work on the same subject, he has reconsidered his view. Another factor leading to his change of view was that in a transudate one would expect red cells and lymphocytes leaving the vessels passively to occur in the effusion in about the same proportion as in the circulating blood, whereas one finds them in the pleural effusion in an entirely different proportion, the lymphocyte often predominating. Throughout, he assumes that the small mononuclear cell is the lymphocyte of the circulating blood, in which he would seem to be justified from its occurrence in the exudate in intimate association with other elements clearly hæmatogenous. Pappenheim, in a foot-note to a paper on the plasma cell, seems to take the view that the cell is derived from the subpleural connective-tissue cells. Patella is convinced that the cell is not a lymphocyte or small round cell, but is derived from pyknotic nuclear fragments of the pleural epithelial cells, an opinion in which he seems to stand alone. The question of the nature of the small mononuclear in the exudate is one with that of the nature of the small mononuclear infiltrating the periphery of the tubercle and tissues generally, a discussion of which cannot be entered into here. Nor may I discuss from this work the question of whether the presence of the lymphocyte in the effusion shows a positive chemotaxis, due to the tubercle bacillus, or whether it shows merely that the exudate is tending to chronicity. Ribbert stands for the view that the lymphocyte speaks for chronicity in an exudate. Some of the recent work with experimental sterile pleural exudates produced by aleuronat injections, notably that of Sweet, seems to show that there is a tendency for the proportion of mononuclears in the exudate to increase rapidly rising from 10 per cent in the first 24 hours to 50 per cent approximately in the third and fourth days. The subject is one that needs further work.

From this study of the pleurisy cases I do not pretend to show that all idiopathic pleurisies are tuberculous. That, I believe, can only be shown by a large series of animal inoculations. What this study has shown, however, is that they have an exudate remarkably uniform in cellular content and general characters, which may be assumed to indicate a uniformity in the nature and etiology of the process. This uniform cellular formula, the work of others seems to indicate, is the formula of tuberculous pleurisy.

Clinically considered, the cases of primary pleurisy studied may be divided into two groups:

First. The largest group includes those which may be termed insidious pleuritis. These cases, though giving a history of a more or less acute onset with cough or pain in the side have had so little fever and so few general symptoms that they have kept about until the volume of the exudate has caused marked dyspnœa. The exudate in this group is practically a chronic one, when aspiration is made.

Second. A smaller group including the cases in which the attack sets in with so marked a fever and such severe general symptoms that the patient is forced to take to his

bed at onset. This group consisted of three cases in this series. The fever ran a course resembling that of typhoid fever, and became normal only after the third week. In one case, which for some time was regarded as typhoid, the Gruber-Widal reaction was tried with the blood serum and the exudate, but was negative in both cases, as were also cultures from the same sources.

The cellular formula shows no distinction between these groups of cases, cases from both groups following the general formula as given above. The aspiration of the exudate seemed to have no effect on the course of the fever in either group.

A possible predisposing factor in these cases, beyond the usual one given of exposure to cold or damp weather, would seem to be indicated by the blood count. The red-blood count was uniformly high, often quite a little above 5,000,000 corpuscles per cubic millimeter, while the hæmoglobin determination was as uniformly in the neighborhood of 70 per cent, thus giving a color index of a little less than 0.7. But a single case of the series has come to autopsy, and that a colored man who had a left-sided pleurisy with effusion in 1900 and a right-sided effusion in the fall of 1901 without tubercle bacilli being demonstrable in the sputum at either admission or without intrapulmonary signs. At the post mortem in November, 1902, almost complete obliteration of both pleural cavities was found and also marked tuberculous involvement of the lungs.

SUMMARY OF CASES.

I. Hospital No. 37,206. Male; white; age 32. Onset after exposure with pain in left side followed by high fever and cough. Fever high and continuous, reaching normal only on 25th day. Aspiration of left pleura on 15th day of disease, 1700 cc. of clear straw-colored neutral fluid. Specific gravity, 1.017, culturally negative. Cellular content shows practically no endothelial cells; a few red-blood cells. Polymorphonuclears, 5 per cent; mononuclears, 95 per cent. Blood count, Hb., 78 per cent; R. B. C., 4,200,000; W. B. C., 10,000.

II. No. 38,067. Male; white; age 43. Onset after exposure with pain in left side followed by cough and sharp fever. Fever continuously high, reaching normal on 27th day. Aspiration on 13th day, 200 cc. of clear fluid, Sp. gravity, 1.019, culturally negative. Polymorphonuclears, 1 per cent; small mononuclears, 99 per cent; a few red cells present. Sputum negative to examination for tubercle bacilli. Blood count, Hb. 78 per cent; R. B. C., 5,032,000; W. B. C., 4800.

III. No. 38,700. Male; colored; age 38. Cough for a week, followed by pain in right side, dyspnœa and fever. Fever ranging between 102 and 103 degrees F., slowly declining to normal on 23rd day. Aspiration on 7th day, 600 cc. slightly bloody fluid, neutral, 1.008. Contains polymorphonuclears, 14 per cent; small mononuclears, 86 per cent; many red-blood corpuscles and few endothelial cells, culturally negative; no tubercle bacilli in sputum. Blood count, Hb. 70 per cent; R. B. C., 5,632,000; W. B. C., 8000.

IV. No. 36,393. Male; white; age 25. Onset with pain in right side, dyspnœa, cough. Moderate fever, remittent in type. Aspiration on 7th day, 1140 cc. slightly turbid fluid, alkaline 1.019, culturally negative. Cellular content; polymorphonuclears, 12.2 per cent; small mononuclears, 84.8 per cent; large mononuclears, 3 per cent. Blood count, Hb. 65 per cent; R. B. C., 4,800,000; W. B. C., 5300.

V. No. 37,252. Male; white; age 35. Onset with pain in left side followed by cough. Fever remittent early, later intermittent. Aspiration on 7th day, 1250 cc. slightly blood-stained fluid, alkaline to litmus, 1.014. Cellular content; polymorphonuclears, 15.1 per cent; small mononuclears, 81.1 per cent; large mononuclears, 3.8 per cent. Three endothelial cells seen in counting 500 cells. Several degenerated nuclear masses; red-blood corpuscles numerous. No tubercle bacilli found in sputum. Blood count, Hb. 75 per cent; W. B. C., 75 per cent.

VI. No. 37,242. Male; white; age 67. Patient suffering from carcinoma of the stomach and marked anæmia. Pain in left side after exposure, followed by dyspnœa. Fever intermittent in character. Aspirated 12th day, 240 cc. slightly blood-stained fluid, alkaline, 1.015. Cellular content; polymorphonuclears, 5.1 per cent; small mononuclears, 91.7 per cent; large mononuclears, 3.2 per cent. Eleven endothelial cells seen in counting 500 cells. Nuclear fragments and red-blood cells numerous. Cultures negative. Blood count, Hb. 50 per cent; R. B. C., 3,576,000; W. B. C., 7000.

VII. No. 36,973. Female; colored; age 10. Onset with cough, followed by dyspnœa. Fever irregular. Aspiration on 15th day, right side, 1200 cc. slightly turbid fluid. Cellular content; polymorphonuclears, 5 per cent; small mononuclears, 95 per cent. No endothelial cells found. Many red-blood cells and nuclear masses. Blood count, Hb. 65 per cent; R. B. C., 4,668,000; W. B. C., 8375.

VIII. No. 38,563. Male; white; age 25. Onset with pain in left side; shaking chill and fever, with cough. Fever slight, irregular. Aspirated on 13th day 500 cc. clear fluid, 1.022. Cellular content; no polymorphonuclears seen on slide in counting 500 mononuclears. A few endothelial cells seen. Red-blood cells numerous. Blood count, Hb. 70 per cent; R. B. C., 6,048,000; W. B. C., 4200.

IX. No. 36,643. Male; colored; age 39. Pleurisy with effusion, left side, six months before, since which symptoms suggestive of pulmonary involvement. Date of onset of right-sided pleurisy indefinite; duration probably some weeks. Fever moderate, intermittent. Aspiration, right side, 375 cc. slightly blood-stained fluid. Cellular content; polymorphonuclears, 3 per cent; small mononuclears, 97 per cent; many red-blood corpuscles. No tubercle bacilli found in sputum. No definite pulmonary signs. Blood count, Hb. 72 per cent; R. B. C., 5,760,000; W. B. C., 11,000. Death November, 1902, one year after pleurisy on right side. At post mortem both pleural cavities obliterated; adhesive pericarditis and tuberculosis in both lungs found.

X. No. 38,141. Male; white; age 21. Onset with pain in right side six weeks before admission. Cough and dyspnœa main symptoms. Fever moderate, intermittent. Aspirated, right side, 980 cc. clear greenish-yellow fluid, 1.021. Ten days later aspirated 1050 cc. clear fluid, 1.019. Cellular content; polymorphonuclears, 2 per cent; mononuclears, 98 per cent, of which small mononuclears are 90 per cent and large mononuclears 10 per cent. A few endothelial cells and red-blood cells found. Blood count, Hb. 70 per cent; R. B. C., 5,736,000; W. B. C., 5100.

XI. No. 38,486. Male; colored; age 43. Onset with pain in back, with some dyspnœa; no other symptoms. Fever intermittent. Fluid examined taken from fourth aspiration of left side on 16th day, 1600 cc. removed, slightly turbid fluid, 1.016. Cellular examination shows a few red-blood cells and endothelial cells. Otherwise sediment is made up entirely of small mononuclears; no polymorphonuclears found on slides. Sputum examination for tubercle bacilli negative. Blood count, Hb. 78 per cent; R. B. C., 4,352,000; W. B. C., 5400.

XII. No. 38,091. Male; colored; age 17. Onset with pain in left side, symptoms few and slight. Fever remittent, showing

rather sharp evening rise. Aspirated left side in third week of disease, 400 cc. slightly turbid fluid, 1.019, neutral. Cellular content slight; red cells few in number. Other cells; entirely small mononuclears. Sputum negative for tubercle bacilli. Blood count, Hb., 58 per cent; R. B. C., 4,784,000; W. B. C., 5250.

XIII. No. 39,039. Male; white; age 9. Onset with cough. Symptoms few. Fever very slight, irregular. Patient aspirated before admission to hospital. Second aspiration some weeks after onset of disease, 30 cc. clear fluid obtained. Fluid shows no polymorphonuclears; red-blood cells plentiful. Sediment made up almost entirely of small and large mononuclears in proportion of 2 to 1. Blood count, Hb. 70 per cent; R. B. C., 4,784,000; W. B. C., 4400.

XIV. No. 38,918. Male; white; age 17. Onset insidious. Patient entered hospital with signs of involvement of lungs, pleuræ, pericardium, and peritoneum. Fever moderate, irregular. Aspiration of left pleura some weeks after onset gave one liter of brownish-red turbid fluid, 1.017. Sediment made up entirely of mononuclears and red-blood corpuscles. Tubercle bacilli found in sputum ten days after aspiration. Blood count, Hb. 68 per cent; R. B. C., 4,492,000; W. B. C., 10,000.

XV. No. 38,614. Male; colored; age 10. Cough week before admission. Chill three days before; otherwise no marked symptoms. Fever remittent with sharp evening rise. Right side aspirated three days after chill, 500 cc. blood-stained fluid, 1.018, neutral, culturally negative. Sediment shows many red cells, few white cells, entirely of mononuclear variety. Blood count, Hb. 71 per cent; R. B. C., 5,492,000; W. B. C., 5330.

XVI. No. 37,858. Female; white; age 58. Cardiac case. Onset with cough, pain in right side and bloody expectoration; dyspnœa and sharp rise in temperature. Aspiration on 8th day, 600 cc. dark blood-stained fluid, 1.017, neutral reaction. Sediment shows many red-blood cells; of the white cells polymorphonuclears are 46 per cent, with small mononuclears 54 per cent. Cells somewhat disintegrated and differentiated with some difficulty. Patient died on following day. No post mortem obtained.

XVII. No. 37,370. Male; white; age 37. Onset with chill and pain in right side after exposure. Sputum slightly blood tinged. Practically no fever. Leucocytes, 9500. Aspiration on 8th day, 260 cc. clear fluid obtained, 1.018, alkaline. Cellular content; large endothelial cells form almost a half of the exudate. Polymorphonuclears, 55 per cent; small mononuclears, 45 per cent; many red-blood cells. No tubercle bacilli in sputum.

XVIII. No. 36,679. Male; white; age 25. Pulmonary tuberculosis recognized March, 1901, when tubercle bacilli were found in sputum. Onset of pleurisy insidious; dyspnœa only symptom. Right side aspirated five times in October and November, 1901, with development of pneumothorax after second aspiration. Cellular content of exudate noted as chiefly polymorphonuclear. Patient returned in March, 1902. Pneumothorax still present. Liter of greenish-yellow seropurulent fluid removed, faintly alkaline, 1.021. Cells somewhat disintegrated. Polymorphonuclears apparently 84 per cent; small mononuclears, 16 per cent. Endothelial cells not infrequent. Tubercle bacilli found in exudate.

XIX. No. 37,138. Male; colored; age 20. Tuberculous symptoms for nine months; pain in right side for two months; definite signs of pneumothorax with fluid. Aspiration, right side, gave 1300 cc. greenish seropurulent fluid, faintly alkaline, 1.025; containing heavy sediment of rather disintegrated cells. Differential count impossible, but polymorphonuclears appear in great majority. Tubercle bacilli found in exudate.

XX. No. 37,037. Male; colored; age 40. Mitral insufficiency with right-sided hydrothorax. Aspiration, 1200 cc. slightly blood-stained fluid, 1.013, slightly alkaline. Cellular content; polymorphonuclears, 1 per cent; large mononuclears, 9 per cent; small mononuclears, 90 per cent. Many endothelial cells and a number of "placards" of three and four cells. Death; post mortem showed no pleurisy.

XXI. No. 36,522. Male; white; age 60. Cardiac, with bilateral hydrothorax. Repeated aspiration of both sides, October 10, 600 cc. from left chest, slightly blood-stained fluid, 1.012. Cellular content; many red-blood cells and small mononuclears. No polymorphonuclears found. Endothelial cells and "placards" numerous. November 4, aspiration right side, with similar fluid and similar cellular content.

XXII. No. 38,362. Female; white; age 17. Chronic nephritis following scarlet fever. Marked œdema, ascites, hydrothorax. March 26, chyloid fluid removed by aspiration from abdominal cavity, 1.010; March 27, left pleura aspirated and 800 cc. of watery fluid with very slight milky tint removed; Sp. gravity, 1.005. Cells form but slight sediment on centrifugalization. A few endothelial cells found and a few "placards," some of four and five cells. Of other cells, small mononuclears form 96 per cent; polymorphonuclears, 4 per cent.

XXIII. No. 37,413. Male; white; age 21. Lobar pneumonia, followed by empyema of left pleural cavity. Exudate showed cells much disintegrated, and almost entirely polymorphonuclears, yet some perfectly preserved small mononuclears found in the proportion of about 4 per cent. The micrococcus lanceolatus was found on cover-slip.

In conclusion I wish to express my thanks to Dr. Osler and Dr. McCrae for their interest in the work, and to my fellow house officers of the medical staff for their kind co-operation.

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A CASE OF PEPTIC ULCER IN THE JEJUNUM OF A DOG FOLLOWING GASTRO-ENTEROSTOMY, WITH A REVIEW OF THE CASES REPORTED IN MAN.[1]

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During the past winter, while Dr. Sowers and I were conducting some experiments upon animals, we had occasion to do a gastro-enterostomy in a dog.

The anterior, or rather the ventral, operation was done. A loop of jejunum, at a distance of about thirty centimeters from the pyloric orifice, was sutured to the ventral surface of the stomach near its greater curvature, care being taken to have the peristaltic movements of the jejunum and stomach correspond in direction.

No mechanical appliance was employed, but after suturing the peritoneal surface of the intestine to that of the stomach by a posterior continuous suture of fine silk, corresponding incisions were made into stomach and intestine, and the cut edges then sutured together around the entire opening by means of a continuous suture of the same material passing through all the coats of each. The anastomosis was completed by a row of mattress sutures around the anterior border of the opening. The posterior continuous suture extended several centimeters beyond either end of the anastomotic opening to prevent spur formation in the intestine.

The animal recovered rapidly and seemed quite well until three months after the operation, when he became sick, refused food, and soon died. At the autopsy death was found to be due to general peritonitis resulting from a perforation of the jejunum near its attachment to the stomach.

On opening the intestine two ulcers were found almost directly opposite to the opening into the stomach. They were in that portion of the intestine distal to the opening, namely, that which would receive the stomach contents and were in no way connected with the line of suture, though the silk suture in places projected through the mucosa and would seem to form a *locus resistentiae minoris*.

The edges of the ulcers were fairly sharply cut and there was little inflammatory reaction about them. In one the process had extended down to the muscular coats of the intestine and in one place to the serosa. The other had perforated and caused death as mentioned above.

The accompanying drawing of the alcoholic specimen will explain itself.

I had the pleasure of showing my specimen to Prof. Mikulicz on his recent visit to Baltimore. He said it was the first instance of the production of such ulcers in animals. I have been able to find no similar case reported.

Since Braun in 1899 first described the formation of peptic ulcer in the jejunum of man, the subject has received considerable attention among German surgeons, but I have been able to find no cases in the English literature.

Cases have been reported by Braun, Hahn, Kausch, Körte, Kocher and others. Altogether I have been able to find fourteen cases reported. This number does not include Hadra's case. See foot note.

REMARKS.

The occurrence of such ulcers in the jejunum after gastro-enterostomy brings up several interesting points for consideration: namely, their frequency, the symptoms produced by them, the factors concerned in their formation, and the influence of their occurrence upon the choice of operation.

I. Their frequency.—The only statistics I have been able to find are those of Kausch, who in 1900 reported two cases among 160 gastro-enterostomies in the service of Prof. Mikulicz. They are probably of more frequent occurrence than these figures would indicate.

II. Symptoms produced by them.—The symptoms of jejunal ulcer are very similar to those of peptic ulcer of the stomach. The burning pain in the epigastrium is usually severe. It is temporarily relieved by taking food but returns with greater severity in an hour or two. Vomiting occurs frequently. Perforation of the ulcer, in case the anterior operation is done, often leads to the formation of a localized abscess, which can be felt as a mass beneath the recti.

III. The factors concerned in their formation.—It seems conclusive that the main factor in their production is the hyperacid gastric juice, which is emptied directly into the jejunum.

In each of the fourteen cases, which I have been able to collect, the operation was performed either for gastric ulcer

[1] Read before the Johns Hopkins Hospital Medical Society, June 1, 1903.

or benign pyloric stenosis, with or without ulcer, and in ten of these there was an hyperacidity of the gastric juices. The giving of bicarbonate of soda after gastro-enterostomy for gastric ulcer has therefore been suggested.

Nevertheless, the very fact that in some of the reported cases the acidity was normal or decreased makes the existence of other determining factors necessary. Some of those which might be suggested are:

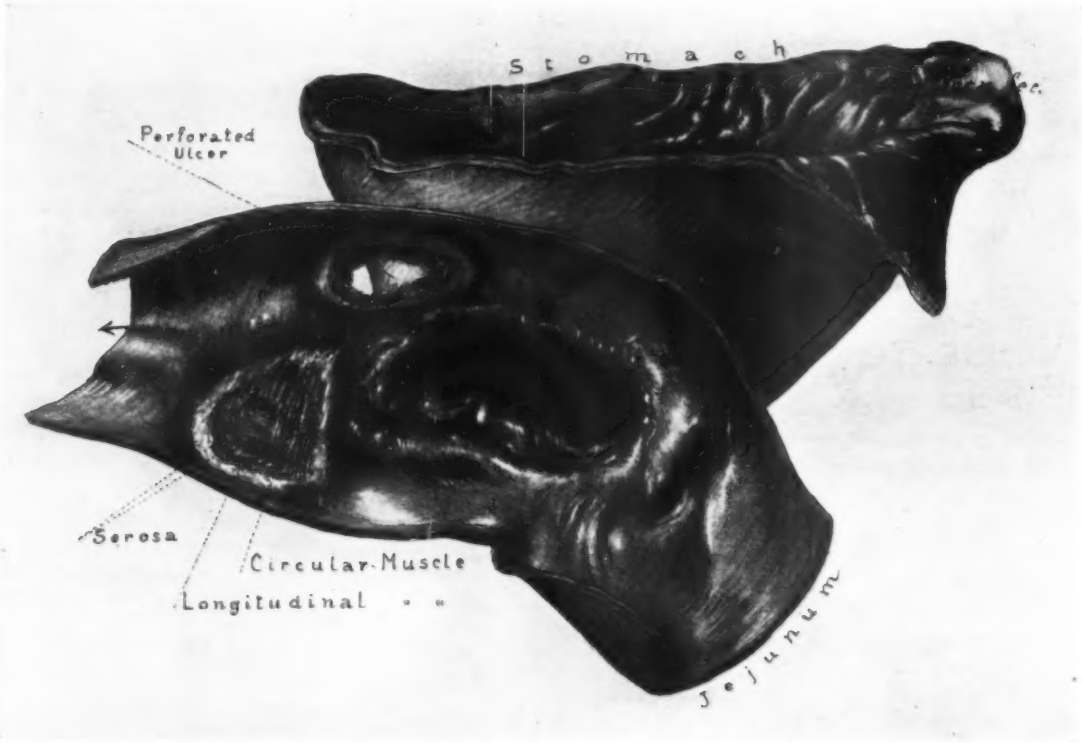
(1) An injury to the mucosa of the intestine by bread crumbs, pieces of bone, foreign bodies, etc. At autopsy I found in the dog's stomach several large stiff straws, which might readily have caused such an injury.

(2) Interference with the circulation of the bowel at the time of operation. Steinthal thinks this happened in his case, which died ten days after the operation; however, the fact that in all the other cases reported the symptoms of ulceration did not appear until much later (three months to three years), would probably rule out this factor.

(3) Traumatism to the abdomen must be considered. In one of Hahn's cases the ulcer followed a blow upon the abdomen. This may have been merely a coincidence.

(4) Kocher thinks the acid gastric juice may stimulate circular contraction of the jejunum just below the stomach with the formation of a kind of cul-de-sac, where the contact of the gastric juices with the intestinal mucosa may be prolonged and give rise to ulceration. He claims to have seen such contraction several times in cases of gastro-enterostomy which he explored.

IV. The influence of their occurrence upon the choice of operation.—The occurrence of these ulcers after gastro-enterostomy is an additional argument for pyloroplasty, rather than gastro-enterostomy in cases of benign stricture of the pylorus, where the former is feasible. The pyloroplastic operation recently devised by Dr. Finney seems applicable to most cases of benign stricture.



Although peptic ulcers do occur in the duodenum, one would naturally suppose its resistance to the action of the gastric juices greater than that of the jejunum; at least, no case of ulcer of the duodenum after pyloroplasty has been reported.

If pyloroplasty is not feasible and gastro-enterostomy is to be done, the question arises as to whether, from our point of view, the anterior or posterior operation is preferable. Of the fourteen cases of jejunal ulcer, which I have collected, the anterior operation had been done in ten, the posterior in two, and the Y operation of Roux in one. In the remaining case the character of the operation is not stated. This seems to indicate that they occur much more frequently

with the anterior operation, but we must remember that the anterior operation has been performed more frequently than the posterior.

If they occur equally often with either operation, then the anterior is to be preferred, for in these cases the jejunum very readily becomes adherent to the anterior abdominal wall, so that perforation of the ulcer often gives rise to localized abscess, rather than general peritonitis. A review of the reported cases proves this to be true. Of the ten cases, in which the anterior operation was done, in three perforation was followed by general peritonitis, and in seven by localized abscess. In both cases in which the posterior operation was done, perforation was followed by general peritonitis.

Kocher's case is the only one of those reported, in which the Y operation of Roux had been done. An ulcer was found in the jejunum just below its junction with the stomach. As Kocher himself remarks, one would very naturally think patients upon whom this operation had been performed especially liable to such ulcers for in them the gastric juice, as it passes into the jejunum, is not neutralized by the alkaline bile and pancreatic juice, which enter the intestine at a lower level.

The whole subject of peptic ulcer of the jejunum is an extremely interesting one and offers an inviting field for further experimental work. I hope to pursue the subject myself.

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ANALYSIS OF REPORTED CASES.

Reference.	Operation, Anterior or Posterior.	Gastric Acidity.	Operation for.	Number of Cases.	Number of Ulcers.	Time after Operation.	Result.
Braun.	Posterior.	Normal.	Dilated stomach.	1	1	1 year.	Perforation; peritonitis; death.
Hahn.	All anterior.	All cases. +	Benign stenosis of pylorus.	3	....	....	1st case, perforation and death. 2d and 3d cases, abscess; operation; cure.
Kausch.	All anterior.	2 cases. + 1 case normal.	1st and 2d, pyloric stenosis. 3d, ulcer of stomach.	3	1st and 2d cases, 1 each. 3d case, ulcer recurred.	1st case, 4 months. 2d and 3d cases, ?	1st and 2d cases, perforation; abscess; operation; cure. 3d case, recurrence of ulcer; jejunostomy; death.
Körte.	Anterior.	+	Gastric ulcer.	1	1	3 years.	Perforation; peritonitis; death.
Steinthal.	Posterior.	+	Pyloric stenosis.	1	4	10 days.	Perforation; death.
Hadra.*	?	+	Pyloric stenosis.	1	1	6 months.	Perforation; abscess; operation; fistula.
Neumann.	Anterior.	+	Functional. Pyloric stenosis.	1	1	3–4 months.	Perforation; abscess; operation; cure.
Kocher.	Y of Roux.	—	Gastric ulcer.	1	1	12 months.	Perforation; abscess; operation; cure.
Heidenhain.	Anterior.	Normal.	Pyloric stenosis with ulcer.	1	1	3–4 months.	Perforation; adhesions; operation; cure.
Goepel.	Anterior.	Both. +	Pyloric stenosis. Gastric dilatation.	2	1 each.	4 months. 13 months.	1st case, perforation; death. 2d case, perforation; suture; cure.

*He is not certain whether ulcers were in stomach or jejunum.

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THE ADVANTAGES OF THE SIMS POSTURE IN CYSTOSCOPIC EXAMINATIONS.

By John A. Sampson, M. D.,
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A full account of the methods used in this hospital, for cystoscopy in women, may be found in Kelly's Operative Gynecology, published by D. Appleton & Co., New York, 1898.

The fundamental principles of a cystoscopic examination have been thus stated by him (see pp. 274 and 275, Vol. 1);

"1. The introduction of a simple cylindrical speculum into the bladder.

2. The atmospheric distention of the bladder induced solely by posture.

3. The illumination and inspection of the vesical mucosa, either by means of a direct light, such as a little electric lamp attached to the forehead or the mouth of the speculum, or by means of a strong light reflected by a head mirror.

The view of the bladder obtained in this way is a direct one; and the open speculum allows the operator to touch any part of the bladder with a sound, and to introduce various instruments with ease."

In regard to the posture of the patient, Dr. Kelly states, "Two postures are available, an elevated dorsal and a knee-breast. The dorsal position is the most convenient to use and the least tiring to the patient, but it is only of service in thin patients, and the atmospheric expansion is not so good; the bladder of a fat women will rarely distend at all in this posture." . . . . . "The knee-breast position is the one position most satisfactory and applicable in all cases."

The advantages of the above method over others where a closed cystoscope is used in the bladder distended with air or fluid are:

1. Various operations may be done through the open cystoscope; as, foreign bodies may be removed, small growths excised, ulcers curetted, etc.

2. Local applications may be made to portions of the bladder, as an ulcer may be treated with silver nitrate, etc., without touching the rest of the bladder.

3. Bloody or purulent urine does not obscure the field of vision as it would in a bladder filled with fluid.

4. The catheterization of the ureters is accomplished under more aseptic conditions when the catheter passes into the ureter through a bladder distended with air than when distended with fluid.

5. Knowledge of the condition of both kidneys may be obtained without catheterizing the ureters; namely, by catching the urine as it comes from the ureter, by pressing a cystoscope with a bevel edge around the ureteral orifice or by means of an instrument devised by Dr. Kelly for this purpose.

6. Also in a known unilateral infection, the diseased kidney may be catheterized and the bladder washed out, and the urine collecting in the bladder will be from the kidney not catheterized, while that from the diseased kidney will pass out of the renal catheter without entering the bladder. Thus we may obtain urine from the supposedly healthy kidney without the danger of catheterizing it in the presence of infection.

7. Bladders of patients with vesico-vaginal fistulæ may in many instances be satisfactorily examined.

8. The instruments used are simpler, less expensive and less likely to get out of order.

The disadvantages of this method are:

1. The knee-breast posture, the one most available and the one which has been used almost exclusively in this hospital, is difficult for a well woman to properly assume and maintain, and still more difficult or even impossible for an invalid or a patient just recovering from an operation.

2. In a very small percentage of the cases the bladder will not distend properly unless a general anesthetic is used.

3. The knee-breast posture is rather an embarrassing one, but on account of its advantages does not deserve the criticisms made by Morris, on p. 324, Vol. II, Surgical Disease of the Kidney and Ureter, Cassel & Co., London, 1901, who makes the following objections to ureteral catheterization:

"1. The obnoxious nature of the operation in the female and the extreme difficulty of it in the male.

2. The unreliableness of the information it affords.

3. The risks to which it exposes the patient.

4. The disadvantages of it as a mode of treatment."

He treats each of these objections separately, and under the first he states, "*The positions in which women are placed,* whether the elevated dorsal or the knee-pectoral, as shown by Howard Kelly's plates, are very revolting to the finer feminine sense, unless the patient is under an anesthetic, and suffice in some instances to make her refuse to submit to a second examination; and as the time required for catheterization of the ureter is often considerable, and when the operation is adopted as a means of treatment for ureteral stenosis or irrigation, it has to be repeated every few days, and sometimes over several months, general anesthesia is out of the question." I might also add that the other objections of Morris to ureteral catheterization are certainly not in accordance with our experience in this hospital.

On April 15, of this year, I wished to examine the bladder of a woman, who had a large post-operative vesico-vaginal fistula. The fistula had resulted from hysterectomy for cancer of the cervix of the uterus, where it had been necessary to resect a portion of the bladder wall in order to get well outside of the growth. The patient was very fleshy and weak, and I realized that she was unable to assume the knee-breast posture. The elevated dorsal, whether the elevation is accomplished by placing pads under the hips as described by Kelly, or by elevating the end of the table, as reported by Webster, Jour. Amer. Med. Ass., 1902, XXXVIII, 1299, did not appear to be applicable in this case. I consequently placed her in the Sims position with buttocks at the end of

the table and elevated slightly the end of the table. A No. 10 cystoscope was used and I found that the bladder dilated well. I could see all parts, both ureteral orifices, and found that the fistula which was oval, had a long diameter of 4 cm., and was situated for the most part posterior to the trigonum, both ureteral orifices being about 1 cm. lateral to the anterior portion of it. The fistula was closed through the vagina ten days later without either a local or a general anesthetic. The bladder was first dissected free and closed and then the vagina was sutured over it. The operation did not cause any pain. The previous operation, namely, the hysterectomy with removal of the upper one-fourth of the vagina, had apparently caused the loss of sensation of pain to the vagina and that portion of the bladder.

From April 15 until the present time, June 18, I have used the posture in 20 patients, and several of these patients had cystitis, requiring local applications, and were treated two to three times a week. I found that in these patients the exposure of the bladder was usually as good, and the ease in making local applications and catheterizing the ureters as great, as in the knee-breast posture. In several instances I examined the same patient in both postures and so was able to compare the two. Of these 20 cases, 5 had vesico-vaginal fistulæ, 6 cystitis, and in the rest the bladders were examined or the kidneys catheterized for diagnostic purposes.

In the cases with vesico-vaginal fistulæ the bladders dilated well in 4 of the cases, and I had no trouble in seeing the ureteral orifices, most important anatomical points to determine before closing the fistulæ. In the case of vesico-vaginal fistulæ where I was unable to make a satisfactory examination, there was a stricture of the urethra, so that I had to use a No. 7 cystoscope, and it caused the patient so much pain that an examination in both positions failed. As stated in the cases where local applications were made for cystitis or the bladder examined for diagnostic purposes the exposure was all that could be desired.

The method of examining the patient is as follows:

1. The patient's bladder must be empty. Either have the patient urinate, which is the better way, or else catheterize her in the dorsal position.

2. The patient assumes the Sims posture on the table, or if a very ill patient, on the bed, i. e., she lies on her left side with both knees and thighs flexed and the right thigh drawn well up above the left; her left arm should be back of her, either hanging over the edge of the table or lying on it, parallel with her back. Her chest should be inclined forward so that she rests upon it. The buttocks must be at the edge of the table or bed.

All the details in this posture must be observed. Both thighs must be flexed, the left as well as the right, and the right thigh must be flexed more than the left in order to permit an exposure of the urethral orifice and also to increase the inclination of the pelvis.

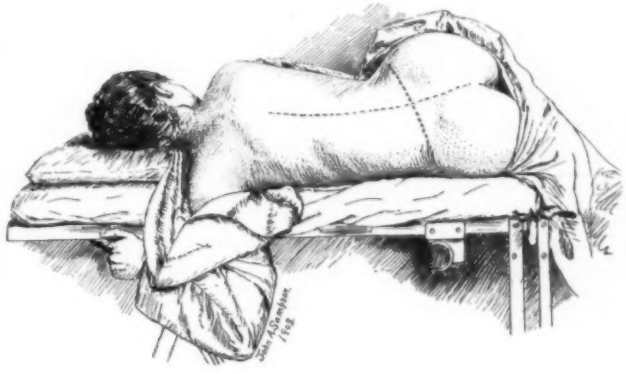
By referring to the illustration which was drawn from a photograph, one can see the effect of this posture on the pelvis.

a. The pelvis is tilted forward as shown by the line drawn across the back from the top of the two iliac crests, and as also shown by the curve in the vertebral column.

b. The pelvis is at the same time elevated.

The effect of the tilting forward of the pelvis with elevation is that the pelvic contents tend to fall out and so we have the same conditions present for atmospheric distention of the bladder, vagina and rectum as in the knee-breast posture, only not as great. In many instances, further elevation of the buttocks is not required.

3. The urethral orifice is now exposed by using two cotton pledgets one on each labia to hold them apart, thus preventing slipping and making the cleansing of the orifice easier. The urethral orifice is cleansed with pledgets which have been boiled in water—use ten pledgets and wipe dry with a sterile dry one. Inject a few cc. of a 10 per cent solution of cocaine into urethra. Leave a pledget in contact with the urethral orifice until ready to examine bladder.



ILLUSTRATION, MADE FROM A PHOTOGRAPH, SHOWING EFFECT OF SIMS POSTURE ON THE PELVIS.

The pelvis is tilted forward as shown by the line drawn across the back from the top of the two iliac crests, and is also shown by the curve in the vertebral column. At the same time the pelvis is elevated slightly. The effect of the tilting forward of the pelvis with elevation is that contents of the pelvis tend to fall out and we have the same condition present for atmospheric distention of the bladder, vagina, and rectum as in the knee-chest posture, only not as great. In many instances, further elevation of the hips is not required. If greater distention of the bladder is desired, it may be accomplished by placing pads under the hips or better by elevating the end of the table.

4. First place a speculum or spatula into the vagina and let that dilate. This is very important no matter whether the patient is examined in the Sims or knee-breast posture. The dilatation of the vagina keeps the trigonum of the bladder from dilating backward when the bladder is examined, and so permits a much better inspection of it.

The urethra is now exposed, dilated if necessary, and the cystoscope is inserted. On removing the obturator the bladder will dilate and its entire mucosa can be examined as in the knee-breast posture. In many instances it is not necessary to elevate the hips, but should the bladder not dilate sufficiently this may be accomplished by placing pads beneath them, or better by raising the end of the table. Thus any degree of dilatation of the bladder possible in a postural distention can usually be obtained.

For physicians who are more dextrous with their right hand the above position is preferable; on the other hand, for those who are left-handed the patient can assume the same posture lying on her right side. As the left urethral orifice is usually seen more readily than the right when the patient assumes the left lateral posture, so the right one will be seen under the same advantages when she assumes the right lateral position.

Judging from the cases which I have examined in this posture, it would seem that it usually afforded the operator all the advantages of the knee-chest posture, a few additional ones, and was free from the disagreeable features of the other.

1. It is not only an easy posture for the patient to assume, but it can also be maintained for a long time without discomfort, a feature which is not only important in the treatment of ill patients but also of those who are in other respects strong and well.

2. The position is a passive one. The patient assumes or is placed in it and once in it does not have to be taught "to bend her back in."

3. The position seems to me to be the least obnoxious one the patient could possibly assume for such treatment.

4. There is less exposure of the patient; the opening in the sheet may be very small and the patient may be completely covered except for this opening.

5. In many cases elevation is not required. The posture, by tilting the pelvis and at the same time elevating it slightly, permits of enough distention of the bladder for a satisfactory examination. Should any elevation be necessary the head can be elevated at the same time, thus obviating in all instances, except where extreme elevation is required, the disagreeable features of the head being lower than the body.

6. Any degree of distention of the bladder possible in a postural distention can usually be obtained by elevating the hips either by placing pads under them or better by elevating the end of the table. When the patient is not elevated the bladder does not distend as much as in the knee-breast posture, a very desirable feature for the trigonum can be the more readily inspected, as well as other portions of the bladder. On the other hand, by elevation one can generally get as great a distention as in the other posture.

7. If for any reason a general anesthetic is required, it is much easier to administer it to a patient in this position than in the other.

8. The exposure of the ureteral orifices varies in different cases. In one group of cases the long axis of the pelvis seems to bisect the trigonum. When it does so the left ureteral orifice is usually seen as easily as in the knee-breast posture and the right or upper one not quite as well. In a second group the trigonum has apparently been drawn down, making the right orifice nearer the long axis of the pelvis than the left. In such instances both ureteral orifices may be seen as readily as in the other position. There is still a third group and that is where the trigonum has apparently been raised and one sees the left ureteral orifice nearer the long axis of the pelvis than the right. In such cases the left ureteral orifice is seen more readily than in the other two conditions, but the right is more difficult to expose. The most extreme case of this kind which I have seen, is in a patient with right pyelitis following pregnancy. I have catheterized the right ureter twice and irrigated the pelvis of the kidney, and plan doing so twice a week until either the pyelitis clears up or other measures are indicated. While the exposure of the orifice is a little awkward, nevertheless I have no trouble catheterizing the ureter. Since the patient is weak and it would be very difficult for her to assume the knee-breast posture, I have not had an opportunity to compare the exposure of the right ureteral orifice in the two postures.

As I have examined only 20 women in this posture I am unable to draw any conclusions as to the relative frequency of the three groups mentioned above.

In the knee-breast posture the exposure of the urethral orifice and thus the introduction of the cystoscope is easier than in the Sims. This advantage is appreciable especially in fleshy individuals, but if one uses cotton pledgets to hold the labia apart, as I have described, one will experience but very little difficulty even in these cases.

There are certain cases where the knee-breast posture gives a better exposure of the bladder than the Sims. I had one such case, a nullipara, twenty-two years old, who was very fleshy. The bladder dilated very little when the patient was in the Sims posture, even when the end of the table was elevated, but on placing her in the knee-breast position a very satisfactory view of the bladder was obtained. The thick abdominal walls relaxed more when she was in the knee-breast posture, thus permitting a greater distention of the bladder. I have examined the bladders of other fleshy women in the Sims posture, and found it satisfactory, but they all had born children and their abdominal walls were more flaccid than in the above mentioned case.

We have then in the Sims posture one which is very easy for the patient, and in most cases satisfactory for the physician, and should an occasional instance arise, as the above, where one cannot obtain a good exposure of the bladder in this posture, the knee-breast or elevated dorsal position may be tried to see if they are better adapted to the individual case. All three postures have their limitations in certain cases.

The knee-breast posture has been the one most generally used in this hospital. Occasionally the elevated dorsal is used in patients who are too ill to assume the knee-breast posture. I have been on the gynecological staff of this hospital since October, 1900, yet in that time I have seen the elevated dorsal position used but twice and never knew of the Sims posture having been used until I used it in April of this year. On making inquiry I find that there have been a few instances in this hospital where patients who were too ill to be held in the knee-breast posture have been placed on their side, their hips were then elevated and thus a cystoscopic examination made.

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